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CONGENITAL PREPYLORIC MEMBRANOUS OBSTRUCTION IN A PREMATURE INFANT*

ARTHUR S. W. TOUROFF, B.S., M.D., AND RALPH M. SUSSMAN, M.D.,
NEW YORK, N. Y.

(From the New-Born Service of the Jewish Maternity Hospital and the Pediatric and Surgical Services of Beth Israel Hospital)

INTRODUCTION

AUTHORITIES[2, 11, 14] agree that, exclusive of hypertrophic pyloric stenosis, congenital obstructions of the stomach are very rare; Pfaundler and Schlossman[11] state that they are so uncommon as to have no special significance. According to von Reuss[14] such lesions, classified by most authors as atresias and stenoses, are found exclusively at the pylorus. Anders,[2] however, states that they may involve the cardia and fundus as well.

Anders[2] is of the opinion that congenital obstructions of the cardia can occur only in the form of septa, similar to those noted occasionally in the esophagus, but cites no cases from the literature. He states that only two cases of congenital occlusion of the body of the stomach have been reported, one by Aregger and the other by Wendel. In the first, obstruction was due to a band of mucous membrane which sprang from the pyloric region in association with hypertrophic stenosis of the pylorus. In the second, the lumen of the stomach was divided by a cystic tumor derived from an accessory pancreas imbedded in the wall of the stomach near the pylorus. Although only a single case of congenital hourglass stomach has been reported (Saake), it is the opinion of Anders[2] that the lesion in question was not of congenital origin. Sternberg and Klose (quoted by Anders) concur in this view and doubt the existence of congenital hourglass stomach. Von Reuss[14] states that he has observed only one case of pyloric atresia, and that Kreuter was able to collect only four cases from the entire literature. Anders[2] emphasizes that, because

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of the difficulty in differentiating at times between acquired and congenital pyloric atresia, the only cases worthy of consideration *as of truly congenital origin* are those encountered during very early life. He states that only three of the latter cases have been reported, two by Hammer and one by Sternberg, and ventures the opinion that they might possibly be explained on the basis of an abnormally high atresia of the duodenum.

Von Reuss[14] divides congenital stenosis of the pylorus into three types. The first is the hypertrophic (Hirschsprung) type, which is too well known to require description. The second is the nonhypertrophic (Landerer-Maier) type, in which the lumen of the pylorus is very narrow but lined by mucous membrane, which is normal except for its thinness. Although this is a true congenital lesion, it seldom produces symptoms during childhood. In most instances the symptoms appear fairly late in life, and operation discloses a pylorus which is narrow without apparent cause. According to von Reuss, only one case of nonhypertrophic pyloric stenosis has been described in an infant. In this case, reported by Schafer, the malformation consisted of a small fistula-like pylorus without a sphincter. The third type of congenital pyloric stenosis mentioned by von Reuss includes connective tissue stenoses and anomalies of formation. These are due to peritoneal bands or adhesions which compress the lumen from the outside and septa of mucous membrane within the lumen. Mya (quoted by von Reuss) believes that the former may originate in fetal peritonitis, while the latter apparently correspond to the congenital occlusions which occur in the remainder of the digestive tract. To von Reuss' three types of congenital pyloric stenosis, a fourth may be added. This is due to compression of the pyloric canal by a tumor, usually a cyst, arising from aberrant pancreatic tissue, imbedded in the wall. Identical in origin with the lesion in the fundus of the stomach reported by Wendel, cysts derived from aberrant pancreatic tissue and occluding the pyloric canal have been reported by Hale,[6] Lyons,[9] Bikoff[4] and others.

Although von Reuss includes septa of mucous membrane in his third group of cases of congenital pyloric stenosis, he cites no cases in which such lesions were found. A personal survey of the literature likewise has failed to reveal a report of such a case. Our interest in this type of congenital occlusion is the result of a recent personal experience in which it was encountered at operation, in a premature infant in whom vomiting commenced at birth. In our investigation of the literature we encountered only one other case in which a barrier of mucous membrane was responsible for obstruction of the pylorus. Although the case bears only slight resemblance to ours, it is of interest. Described by Bonner[5] in 1912, it concerned a 4-week-old infant, who presented symptoms of "intractable pylorospasm" unrelieved by intensive medical treatment. At operation, the stomach was found to be greatly distended and the

pyloric sphincter "unusually developed." A posterior gastroenterostomy was performed, but the child died seventeen days later. At postmortem examination the pyloric sphincter was markedly hypertrophied, but, in addition, the mucous membrane of the greater curvature of the stomach, at the entrance to the pylorus, was duplicated in such a fashion as to form a flap which closed the pylorus completely when attempts were made to force fluid from the stomach into the duodenum. When fluid was caused to flow from the duodenum toward the stomach, however, the mucous membrane flap was forced back into the stomach and thus offered no resistance to the passage of the fluid. The unusual anatomical findings described above explained why bile entered the stomach and appeared in the vomitus in the presence of complete pyloric obstruction. Whether the obstructing flap was merely a redundant fold of mucosa, such as is commonly encountered within the pyloric lumen in cases of hypertrophic pyloric stenosis, or whether it represented a separate congenital anomaly is difficult to state. Since an extensive review of the literature has failed to reveal a case similar to the one about to be reported, it appears warranted to present the latter in detail.

CASE REPORT

B. L., a white female infant, was born at the Jewish Maternity Hospital, New York, N. Y., on Sept. 11, 1938. This date was approximately four weeks in advance of the completion of full term. The mother, a native-born woman, 38 years of age, whose pregnancy was complicated by marked polyhydramnios, went into labor spontaneously and was delivered without difficulty six hours later by one of us (R. M. S.). Her only other pregnancy, seven years previously, also had resulted in the birth of a premature child who suffered continuous cyanosis and died on the third day of life. No post-mortem examination was performed but, in view of the paternal family history about to be presented, the possibility of death from a congenital anomaly must be considered.

The infant's father, who was one of three siblings, had two sisters. Each of the latter bore only a single child. Both of these children died in infancy, one of cerebral agenesis and the other of mongolism. Thus, three and possibly all four offspring (including B. L.) of the patient's father and his two sisters suffered from some congenital anomaly. Accordingly, it is warranted to assume that the developmental defect observed in B. L. was of paternal origin. Another possibly related factor was the existence of marked polyhydramnios during the mother's pregnancy. The frequent association of this condition with congenital anomalies and premature birth is well known, and reference concerning it may be found in practically all standard works on obstetrics.[15]

At birth, B. L. weighed six pounds and presented no gross external abnormalities. Initial cyanosis, however, was quite marked and responded to intensive treatment only after a period of five hours. No meconium was passed during or after delivery. The child was placed upon a feeding formula, which she took well. Within a short time, however, she began to vomit, the vomiting occurring toward the end of each feeding. This regurgitation appeared effortless and was nonprojectile in character. Various changes in the amount and composition of the formula failed to influence the vomiting appreciably, and it soon became evident that no food was being retained. During the first and second days the child had no spontaneous bowel movements, even of meconium; and an enema administered at

the end of the second day resulted in the return of only a small amount of colorless mucus. In spite of this apparent evidence of obstruction of the gastrointestinal tract, the abdomen was not distended. On the third day, because of progressive loss of weight and dehydration and the continuation of obstipation and vomiting, she was transferred to the Pediatric Service of the Beth Israel Hospital. There, the parenteral administration of fluids was begun. In view of icterus, which appeared more marked than was to be expected on the third day of life, the urine was examined for bile and found positive. Examination of the vomitus for bile was negative, as were similar examinations on a number of subsequent occasions.

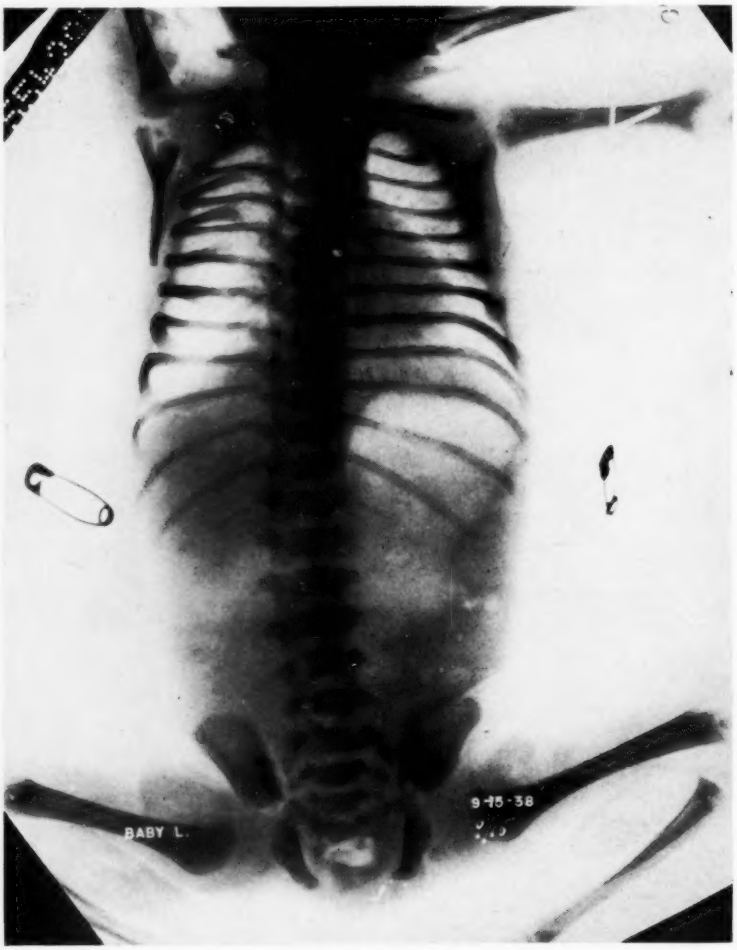

Fig. 1.—Fourth day of life. Stomach diffusely distended. No gas in small intestine. Bubbles in rectum, sigmoid, and splenic flexure are due to air accidentally introduced with previous enemata.

On the fourth day, because of persistent obstipation and vomiting, x-ray examination was performed (Fig. 1). This revealed the stomach to be diffusely distended with air, the body of the viscus extending downward to a point about 1 inch above the level of the iliac crests, and across the midline to the right side of the abdomen. It was uncertain whether or not the first portion of the duodenum formed a part of this air-filled pouch. The small intestine contained no gas; and the few bubbles noted in the rectum and in the sigmoid and splenic flexures of the colon,

therefore, were assumed to be due to air, introduced accidentally with previous enemas. These findings indicated the presence of a complete obstruction, involving either the pyloric portion of the stomach or the very first portion of the duodenum. A film made shortly thereafter (Fig. 2) revealed the structure in question to have become distinctly bilocular. The distal portion now was globular in contour and relatively more distended than previously. It reached down almost to the level of the right iliac crest and was separated from the proximal portion by a well-marked constriction. The findings still were difficult to interpret since it was not clear whether the distal air-filled pouch represented greatly dilated, proximal portion of duodenum, separated from the stomach by the pylorus, or whether it represented a dilated pyloric antrum separated from the body of the stomach by a contraction of the musculus sphincter antri.

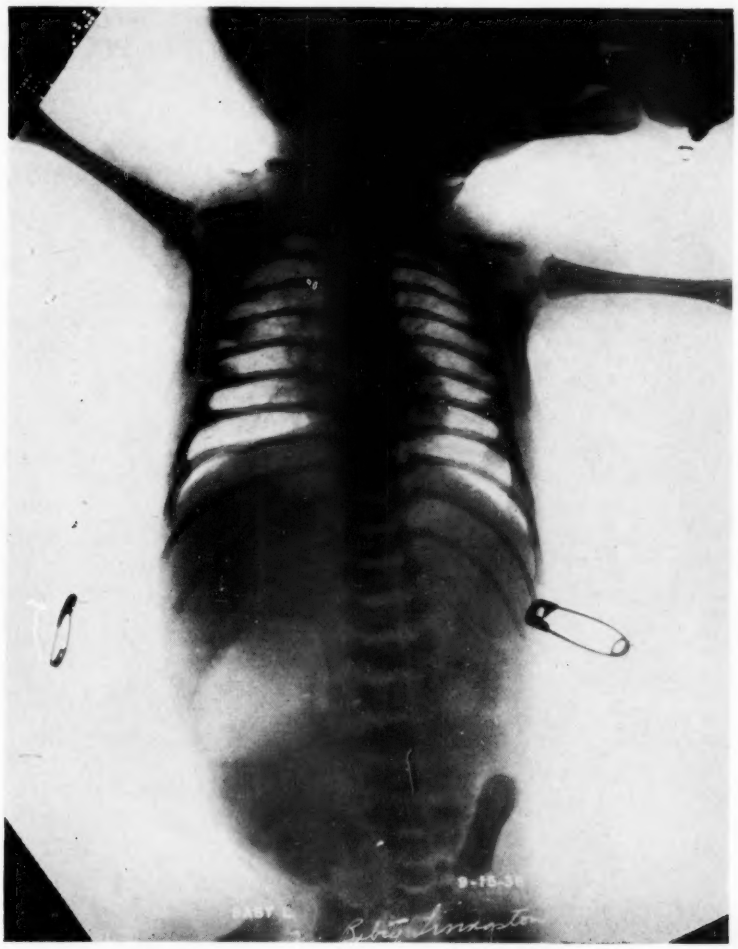

Fig. 2.—Several hours later. Stomach now is bilocular. Distal pouch (antrum) distended and separated from body and fundus by contraction of musculus sphincter antri.

On the fifth day all symptoms continued and the child lost ground steadily in spite of the administration of several transfusions and large quantities of fluid by vein and subcutaneously. At that time the stomach was aspirated, a small amount of thin barium mixture administered by gavage, and x-ray films taken immediately.

In the previously described distal pouch, the opaque medium formed a dense, smooth-edged, ovoid shadow, measuring about 1¼ by ¾ inches. During a short period of fluoroscopy, no barium passed beyond this point. Films made four hours later (Fig. 3) revealed the body and fundus of the stomach to be greatly distended still with air and to contain practically no barium. The dense, ovoid, distal shadow had become larger in size and more circular in contour. Barium still had not advanced beyond this area, indicating conclusively the presence of complete obstruction. Whether the latter involved the first portion of the duodenum or the distal portion of the stomach still was uncertain. However, in view of the persistent obstruction, regardless of its exact site, it appeared warranted to assume that continuation of conservative therapy would be unavailing and surgical opinion, therefore, was requested.

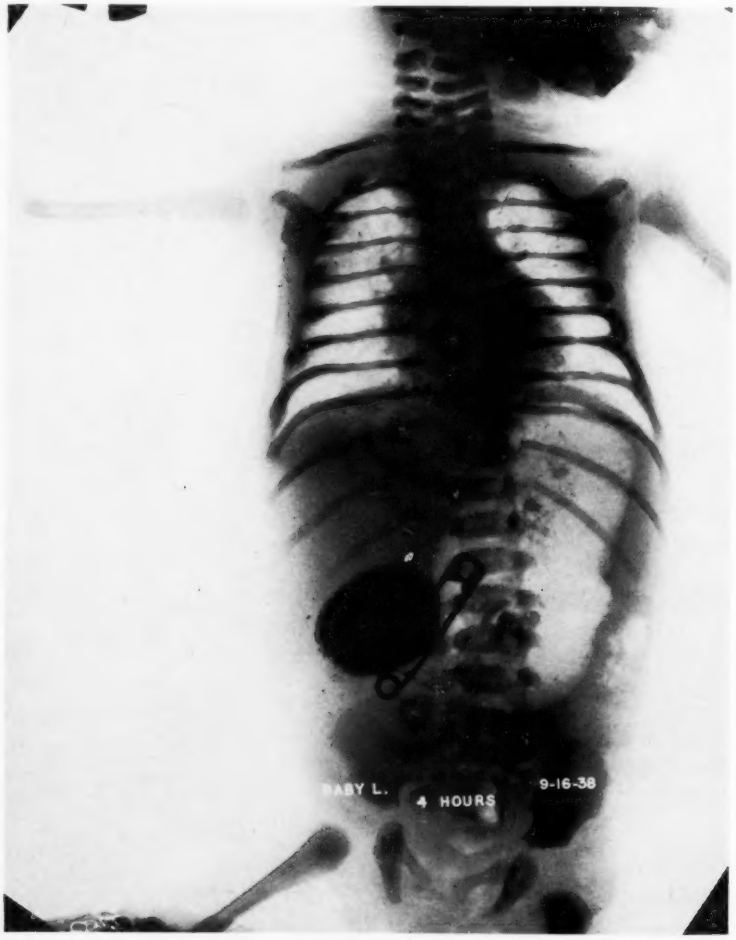

Fig. 3.—Four hours after ingestion of barium. Barium-filled antrum remains sharply demarcated from the air-distended body and fundus by spasm of the musculus sphincter antri. No barium in small intestine, indicating presence of complete pyloric obstruction.

When seen in consultation by one of us (A. S. W. T.) on the afternoon of the fifth day, the temperature was normal. Pulse and respiration were too rapid to count with accuracy. Jaundice was fairly deep, and there was evidence of moderate dehydration. The weight was 5 pounds, 4 ounces (a loss of more than one-seventh of the birth weight). Considerable local swelling and edema were present in the

pectoral, axillary, and scapular regions as the result of poor absorption of previously administered clyses. (Taking into consideration the volume of this retained fluid, the child's weight undoubtedly was less than 5 pounds.) The mouth, tongue, and pharynx were deeply congested and studded with adherent white patches characteristic of thrush. The abdomen was not distended and was soft throughout. No abdominal masses were palpable and no peristaltic waves were visible. During examination the child vomited several mouthfuls of clear, yellowish fluid. Gastric aspiration yielded 40 c.c. of similar fluid which on chemical analysis contained no bile. The blood count was as follows: R.B.C., 4,200,000; hemoglobin, 160 per cent (due to dehydration); W.B.C., 8,900 with 61 per cent polymorphonuclear leucocytes. The urine contained a trace of bile but otherwise was not remarkable. A rectal irrigation administered at the time, was totally ineffectual.

Discussion of Diagnostic Features.—On purely clinical grounds the continued vomiting, obstipation, and ineffectiveness of enemas since birth five days previously warranted the diagnosis of a congenital anomaly resulting in obstruction of the gastrointestinal tract. The absence of bile in the vomitus on all examinations and the lack of abdominal distention appeared compatible with a very high obstruction involving either the stomach or the duodenum proximal to the papilla of Vater. The very early appearance of symptoms, the sex of the child, and the absence of projectile vomiting, visible peristalsis, and palpable abdominal mass constituted strong presumptive evidence against the diagnosis of congenital hypertrophic pyloric stenosis.

The presence of bile in the urine, in conjunction with icterus which appeared more intense than that usually to be noted on the fifth day of life, could be explained in two ways. The first possibility was that they were merely an accompaniment of prematurity. According to Holt and Howland,[7] icterus neonatorum may or may not be present in normal newborn infants. These authors state that in premature and delicate infants, however, it occurs regularly and is apt to be more severe and to last longer than normally. They also emphasize that bile, which ordinarily is not present in the urine in mild cases, is apt to be noted in the more severe cases. The second possibility as regards the significance of icterus and bile in the urine, especially in a patient in whom one congenital anomaly already was assumed to be present, was that these manifestations were due to the presence of an additional anomaly such as an obstructive lesion of the biliary duct system. The latter might be either a stenosis due to external pressure or an atresia at some point in the duct system. Biliary obstruction of either of these types, if complete, would have accounted satisfactorily for the absence of bile in the vomitus. Under such circumstances the occlusion of the gastrointestinal tract, discussed above, might have been situated in the duodenum distal to the papilla of Vater or in the uppermost portion of the jejunum. The lack of abdominal distention, however, made it appear unlikely that the obstruction could be situated distally to the latter point.

The complete absence of the passage of meconium or stool at any time during life could be explained in two ways. The first was by the presence of still another anomaly, an obstruction involving the lower bowel. The second explanation was that offered by Alverez,[1] who pointed out that the best stimulus to intestinal peristalsis is the passage of gastric contents through the pylorus into the duodenum. In accordance with this theory the presence of complete pyloric obstruction alone could account for the failure of our patient to pass meconium.

Roentgenographic examination confirmed the clinical impression of a high, complete obstruction, which, for reasons to be discussed later, appeared to be intrinsic in nature. The exact site of obstruction, i.e., either in the very first portion of the duodenum or in the distal portion of the stomach, was uncertain although the latter appeared more likely. The examination offered no information in regard to the question of an obstruction of the biliary passages and shed no light on the problem of the existence of an additional low intestinal obstruction as seemed quite possible from the failure of the child ever to pass meconium.

Regardless of the presence or absence of anomalies in addition to the high complete obstruction, immediate operation was advised, for the reason that the child was starving in spite of parenteral feeding and death seemed inevitable unless the obstruction were relieved. Because of the patient's prematurity and generally poor physical condition, a bad prognosis was given.

Operation.—On Sept. 16, 1938, under open ether anesthesia the abdomen was opened by one of us (A. S. W. T.) through a 4½ inch right paramedian, muscle-splitting incision, which extended from the right costal arch to a point below the level of the umbilicus (Fig. 4*e*). The stomach, which was greatly enlarged, presented in the wound at once. On exploration it was found to occupy most of the left half of the abdomen, from the diaphragm almost to the level of the iliac crest, and to extend across the midline to the right side. In the latter region its upper half was covered by the free margin of the liver. The latter organ, which was about one-half the size of an adult's fist, was found displaced entirely into the right side of the abdomen by the distended fundus and body of the stomach (Fig. 4). On retracting the margin of the liver upward and laterally and drawing the proximal portion of the stomach to the left, the upper half of the distal portion of the stomach and the first portion of the duodenum were exposed. The edges of the wound then were retracted widely and the major portion of the stomach visualized. The viscus measured about 5 by 2½ inches in its greatest diameters, the fundus and body being demarcated from the antrum by a fixed contraction (spasm) of the musculus sphincter antri which, in effect, divided the organ into two pouches. The proximal pouch, consisting of the body and fundus, measured about 3 by 2½ inches in diameter. Its walls were judged to be of normal, or perhaps slightly less than normal, thickness. It was distended with air and fluid, but could be indented readily. The distal (antral) pouch measured about 1½ by 2 inches in diameter and was roughly globular in shape. Its walls were appreciably thicker and more resistant to pressure than those of the proximal pouch. As the organ was handled, the muscular contraction between the two pouches relaxed somewhat. The duodenum measured about ¼ inch in diameter, its most proximal portion widening slightly as it joined the dilated antrum. The junction of the stomach and duodenum was clearly marked by the pale, glistening pyloric ring. Due

to the dilatation of the antrum, the site of the pyloric ring was further marked by a relative, slight external constriction. As the result of the marked downward and lateral displacement of the enlarged antrum, the first portion of the duodenum had become considerably elongated and coursed upward quite sharply for a distance of about 1 inch to reach the region of the head of the pancreas. The second and third portions of the duodenum were visualized indistinctly.

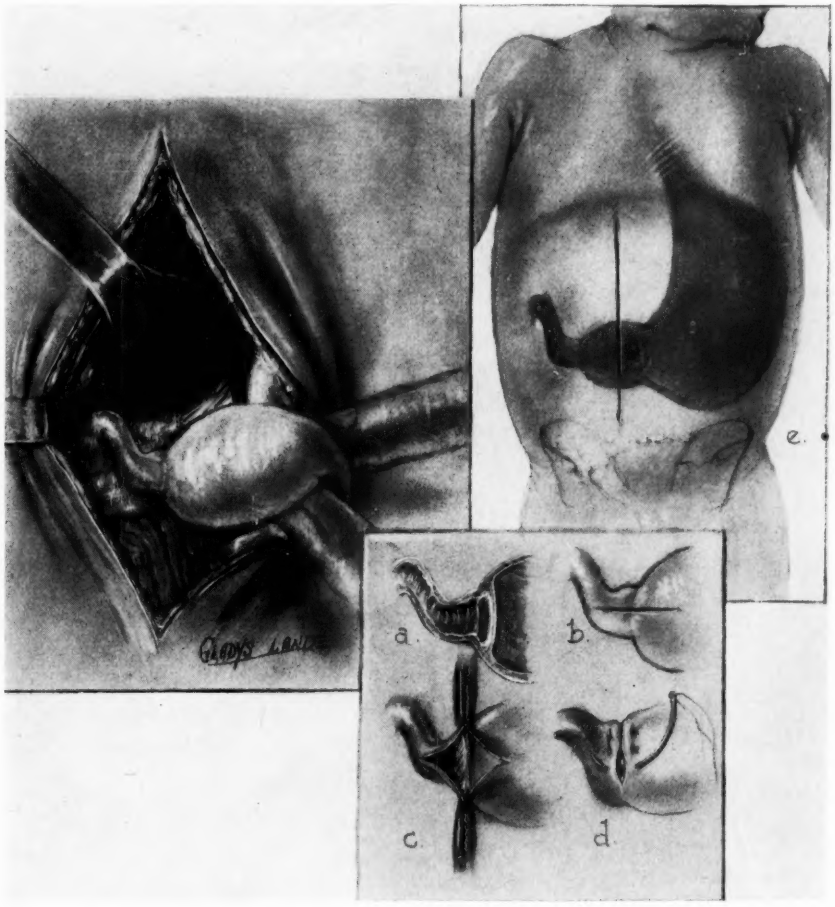

Fig. 4.—Operative findings and procedure. Hypertrophied dilated antrum retracted toward the left, to expose duodenum and gastroduodenal junction. Note sharply demarcated pyloric ring and normal caliber of duodenum. Duodenum is elongated and rises sharply to reach head of pancreas. Entire liver displaced into right side of abdomen by distended stomach. Large bowel almost completely hidden. Small intestine empty and contracted. *a*, Cross section of antrum and adjacent duodenum showing site of mucous membrane septum in immediate prepyloric area; *b*, incision through proximal portion of duodenum and adjacent stomach; *c*, incision about to be closed at right angles to its original direction (Heineke-Mikulicz pyloroplasty); *d*, pyloroplasty continued; inner (continuous) suture completed; outer tier of mattress suture being placed; *e*, diagrammatic sketch of site and extent of incision.

No external peritoneal bands or folds were present at any point on the surface of the stomach or duodenum, thus ruling out external constriction as the cause of obstruction. Since the stomach was distended, the pyloric ring clearly visible, and the duodenum normal in caliber, it was evident that the obstruction was due to an intrinsic lesion situated in the distal portion of the stomach. To prove this point, the proximal portion of the stomach was compressed manually in order to force its contents distally. As this was done, the antrum became tensely bal-

looned with gas and fluid, but the caliber of the duodenum did not change in the slightest degree. The site of obstruction thus having been established at the pylorus, the abdomen was rapidly explored for evidence of other anomalies. The other viscera because of their small size are worthy of brief description. The gall bladder, which contained bile, measured ½ by ¼ inch and emptied readily on pressure. The fact that bile had entered the gall bladder and could be expressed from it without difficulty constituted fairly strong evidence against the presence of an obstructive lesion involving the extrahepatic biliary system, as had been suggested by the icterus and the presence of bile in the urine prior to operation.

The pancreas, which was quite mobile and could be visualized completely, measured about ¾ by 3/16 inch. The spleen measured about 2½ by 1½ inches and was distinctly lobulated. The small bowel, commencing at the fossa of Treitz, was firmly contracted throughout its entire length, its diameter being about 3/16 inch. The large intestine, which was completely hidden by the distended stomach except in the region of the hepatic flexure, also was contracted. It measured about ⅜ inch in diameter and contained a small amount of secretion in its distal portion. The appendix measured about ½ by ⅛ inch. No external, constricting peritoneal bands or folds or areas of atresia were noted involving the small or large bowel. The omentum, which was about 2 inches long, was extremely thin and delicate and of almost gelatinous consistency. It was light brown in color and appeared not to contain fat. No anomalies other than the original obstruction in the pyloric area having been encountered, attention was redirected to that point.

A short incision was made in the anterior wall of the juxtapyloric portion of the duodenum parallel to its long axis, and carried proximally to the pyloric ring. Bile-stained, duodenal secretion immediately flowed from the opening. This indication that the common bile duct was patent together with the evidence presented previously that the gall bladder contained bile and could be emptied on pressure constituted further, but not necessarily complete, proof of the absence of an anomaly of the biliary passages. (In the presence of icterus and bile in the urine, an obstruction in one of the branches of the hepatic duct still was theoretically compatible with the operative findings.) A large blunt probe was passed distally into the duodenum without encountering obstruction. It was then passed proximally through the pyloric ring for a short distance, where an impassable obstruction was met. Several attempts to overcome the obstruction being unsuccessful, the probe was withdrawn and pressure again was made on the proximal portion of the stomach in order to balloon up the antrum. As this was done and the edges of the duodenal incision were held open, a small nipplelike projection was seen to protrude through the pyloric valve and to extend into the lumen of the duodenum for a short distance. As the stomach was still more firmly compressed, the nipplelike projection became distended and more globular. Its appearance now was that of a small balloon which, in part, had been forced from the gastric side of the pyloric ring into the lumen of the duodenum. It was grasped immediately with small forceps and incised with a scalpel. As this was done, gas and fluid escaped at once, and the stomach, the proximal portion of which was being compressed, collapsed. The short duodenal incision, which ended at the pyloric ring, then was continued proximally into the adjacent portion of the stomach for a distance of about ½ inch and the lesion within the lumen exposed. It was found to consist of a complete septum or diaphragm, which lay transversely in the distal portion of the stomach, immediately proximal to the pyloric valve (Fig. 4*a*). Both surfaces of this diaphragm were covered by mucous membrane which merged at the periphery with the mucous membrane of the stomach and the adjacent pyloric valve. The edges of the stab wound previously made in the septum were grasped, and the latter was incised radially in several directions with the result that it retracted peripherally in every direction to such an extent that it was no longer distinguishable from the mucous membrane of the adjacent

gastric wall. In order not to narrow the lumen of the gastroduodenal junction or the duodenum, a Heineke-Mikulicz pyloroplasty was performed by closing the existing horizontal incision in a vertical direction (Fig. 4*c* and *d*). Two layers of sutures were employed for this purpose: an inner, continuous Connell stitch of No. 000 chromic catgut, reinforced by an outer tier of interrupted No. 000 chromic mattress sutures. After the incision had been closed, gas and fluid could be milked readily from the stomach into the duodenum. In order to terminate operation as quickly as possible, the abdomen was closed with interrupted through-and-through sutures of heavy silk, which approximated the structures of the abdominal wall in one layer.

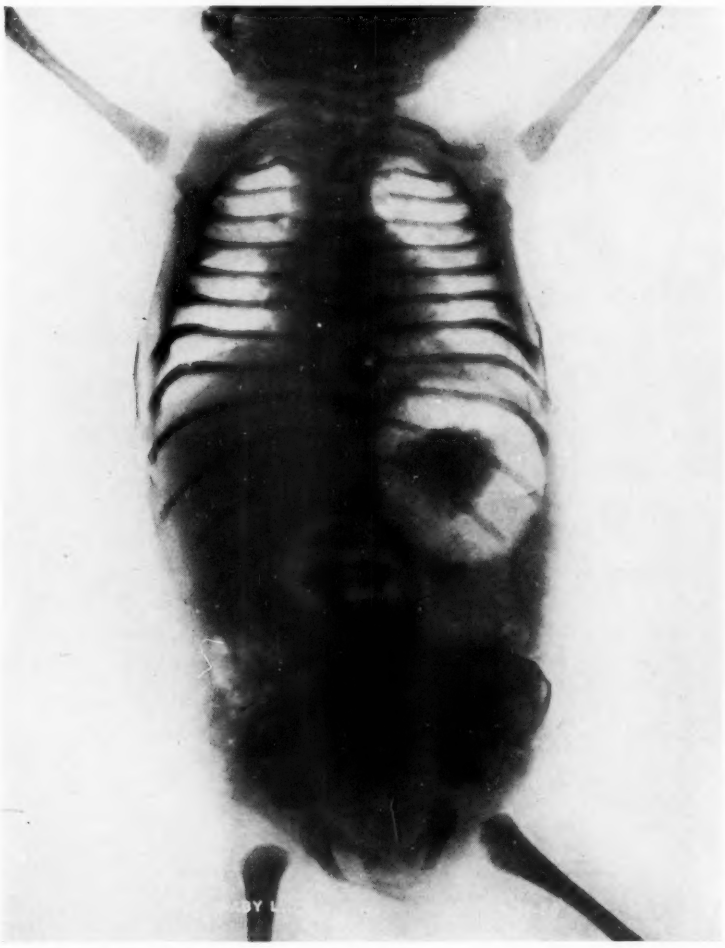

Fig. 5.—Two days postoperatively. Stomach still bilocular, but smaller. Antrum now situated in front of vertebral column. Note gas-filled first portion of duodenum, shaped like reversed letter S, immediately beyond the antrum. Gas also present in intestine below duodenum.

Postoperative Course.—Although the immediate postoperative reaction was satisfactory, a prophylactic transfusion of 60 c.c. of citrated blood was administered the same night. At the end of six hours, feedings of 5 c.c. of glucose in Ringer's solution were given every half-hour. Periodic postoperative vomiting occurred during the first twenty-four hours, but the major portion of the feedings were retained. The vomitus now (for the first time) contained bile, indicating patency

at the operative site. On the day after operation, a small amount of meconium and then stool were passed for the first time. The bowels thereafter moved several times daily. There was some elevation of temperature during the first two days, the peak being 101.8°. By the third day, temperature was normal. Two days after operation, x-ray films of the abdomen were made (Fig. 5). The stomach, which still was bilocular, was found considerably less distended than previously. The antral portion now was situated in front of the vertebral column instead of in the right side of the abdomen. Immediately beyond the antrum, the gas-filled first portion of the duodenum, resembling a reversed letter S, could be clearly visualized. Gas also was present for the first time in other portions of the bowel.

The feeding formula was increased rapidly in amount and was taken without vomiting. Jaundice began to subside and within a week practically disappeared. The wound healed kindly except for the occurrence of slight necrosis of the skin in the central portion. Alternate sutures were removed on the seventh day following operation and the remainder on the twelfth postoperative day. By that time the oral lesions (thrush), which had been treated by the local application of gentian violet, had subsided completely. Over a period of two and one-half weeks the child's weight increased from approximately 5 pounds to 6 pounds 3 ounces, and at the end of that time she was ready to be discharged from the hospital.

General Discussion.—At this point it is of interest to attempt to correlate the roentgenographic and clinical features of the case with the operative findings and postoperative course. In the light of the operative findings, which consisted primarily of a complete, transverse septum of mucous membrane in the prepyloric region, a reconsideration of the x-ray films is of value and may offer some criteria upon which the diagnosis of pyloric occlusion may be based in the future. Interest centers chiefly upon the distal air-filled pouch seen in Fig. 2 and the corresponding barium shadow in Fig. 3. In Fig. 1, although the stomach is diffusely distended, its contour is fairly normal. In Fig. 2 a bilocular organ is seen, the distal pouch being globular in shape and relatively more distended than the corresponding portion of the viscus in the preceding film. Whether this distal pouch represents the first portion of the duodenum or the pyloric antrum is uncertain. Another film (not reproduced in this article) was taken immediately after barium had been introduced into the stomach through a catheter. Under these circumstances, the distal air-filled pouch noted in Fig. 2 became filled with barium at once. This observation at first glance tended to support the view that the pouch represented the distal (antral) portion of the stomach. Another possibility, however, was that it represented the dilated, proximal portion of the duodenum. In order to render the second interpretation tenable, it was necessary to postulate that the pylorus was so atonic as to permit barium to flow from the stomach into the duodenum at once. (According to Morton and Jones[10] and to Ladd,[8] such atony and dilatation of the pylorus occur quite regularly in association with marked duodenal dilatation above a point of obstruction.) After four hours, barium in increased amount had been forced into the distal pouch, while the remainder of the stomach contained practically none (Fig. 3).

If the pouch represented a dilated duodenum associated with a widely dilated, atonic pylorus, the thin barium mixture should have flowed back into the stomach fairly freely and, as a result of vomiting, the shadow under discussion should have become smaller or even disappeared. Instead, it grew larger and maintained its rounded contour. This sequence seemed to indicate the presence of strong muscular contraction in the area proximal to the barium-filled pouch. Since such muscular contraction is incompatible with a dilated, atonic pyloric sphincter, the diagnosis of duodenal dilatation seems unlikely. Even if it were assumed for the purpose of argument that the pouch represented a dilated duodenum separated from the stomach by a firmly contracted pyloric sphincter, the latter would have rendered it impossible for barium to have passed out of the stomach as quickly as it did in the first place. Accordingly, by a process of exclusion, it seems most logical to assume that the pouch represents the antral portion of the stomach separated from the body of the viscus by a contraction of the circular musculature (musculus sphincter antri) at the antral inlet. Schindler,[12] as the result of his gastroscopic observations, recently has drawn particular attention to this structure. A roentgen picture such as has been presented apparently is seen only in the earlier phases of pyloric obstruction when the antral (motor) portion of the stomach has undergone compensatory hypertrophy and moderate dilatation as the result of continued attempts to force the gastric contents beyond the point of obstruction. It is of interest to note that this was precisely the state of the antrum as disclosed at operation.

Although an anomalous obstruction, responsible for the major symptoms, was found in the prepyloric area, none was found in the biliary system to account for the icterus and bile in the urine, and none in the lower bowel to account for the failure of the child ever to pass meconium or to move its bowels prior to operation. The fact that the icterus subsided within a week of operation indicated that it was of the severe neonatal type, which, according to Holt and Howland,[7] often occurs in association with premature birth. The passage of a small amount of meconium for the first time shortly after operation indicated that this symptom, as Alvarez[1] suggested, was due to the complete pyloric occlusion. The stimulus to intestinal peristalsis apparently originates primarily in the passage of gastric contents into the duodenum. Thus it may be assumed that normally a newborn child will pass meconium as soon as gastric secretion passes into the duodenum, even before food is taken. In our patient, since the obstruction in the prepyloric area was complete, no gastric contents passed into the duodenum and therefore the major stimulus was lacking. According to Ballantyne,[3] amniotic fluid, vernix caseosa, lanugo hair, and epidermal cells normally are swallowed during fetal life and pass into the intestinal canal to increase the bulk of the meconium. It appears quite likely that the small amount of meconium noted in the colon at operation and passed by the child after operation was due to the failure of the above-mentioned constituents to pass from the stomach into the intestine during fetal life.

An attempt to explain the presence of a complete septum of mucous membrane situated immediately proximal to the pyloric valve results in speculation. The first theory is that the septum is formed as the result of fusion of folds of mucous membrane which in this region normally are reduplicated to form the pyloric valve. A second explanation is that the septum represents an abnormally high occlusion of the duodenum, as suggested by Anders to explain the three cases of pyloric atresia reported by Hammer and by Sternberg. According to Tandler,[13] prior to the fifth week of gestation the intestine possesses a lumen lined with epithelium. Shortly thereafter the epithelium proliferates and fills the lumen to obliterate the latter completely. This solid stage lasts only a short time, to be followed by a stage in which multiple vacuoles appear. These vacuoles coalesce and by the twelfth week of fetal life the lumen normally is re-established. A failure of complete coalescence of the vacuoles results in the presence of residual areas of atresia or of septa which occlude the lumen, such occlusions being found most commonly in the duodenum. Accordingly, an abnormally high duodenal occlusion might possibly be considered the cause of the anomaly noted in the present case. Unfortunately a specimen of the mucous membrane forming the septum was not removed for microscopic examination, but there was no doubt that the septum was situated proximal to the pyloric ring.

Further Course.—On the eighteenth postoperative day, after sixteen days of normal temperature, there was a rise to 101.4° and the child vomited several times. Examination was negative except for the presence of a moderately severe pharyngitis, and it was assumed that the latter accounted for the fever and vomiting. On the following day, temperature rose to 106.2° and vomiting became more frequent. At that time the pharyngitis appeared more acute. By the third day temperature returned to normal, but vomiting continued and the bowels had failed to move for thirty-six hours. All feedings therefore were discontinued, and fluids again were given parenterally. Enemas at first resulted in the return of small amounts of stool, but subsequently were ineffectual even for gas. During the third day, the abdomen gradually became distended. In the presence of a clinical triad, consisting of persistent vomiting, obstipation, and increasing abdominal distention, the diagnosis of intestinal obstruction was made. The presence of bile in the vomitus and the abdominal distention contrasted with the clinical picture of the original obstruction in which both of these features were absent. Accordingly, it was postulated that the obstruction was not present at the original site, but at a point lower in the small intestine. An x-ray film of the abdomen taken on the fourth day of obstruction revealed the presence of a large fluid level which, because of its size and its position in the left upper quadrant, appeared to be situated within the stomach. Details of the abdominal viscera below the fluid level were obscured, however, by a diffuse haze which involved the entire abdomen. Within this hazy area a number of scattered fluid levels were seen, the largest being situated in the left half of the abdomen below the gastric fluid level mentioned previously. In order to establish the significance of these scattered levels more precisely, the stomach was emptied by tube, a tiny amount of barium introduced, and another film made. This revealed moderate gaseous distention of the stomach, the lower border of which was faintly demarcated by the barium, and disclosed the presence of several distended loops of small intestine containing gas and fluid. The most prominent of these were situated on the left side below and

partially lateral to the greater curvature of the stomach. These findings confirmed the diagnosis of obstruction of the small intestine. Because of the location of the distended loops, in the upper abdomen and chiefly toward the left side, the obstruction was considered to be situated in the jejunum. It was further assumed to have been produced by angulation or compression of the bowel by plastic exudate at or near the operative site.

Temperature had been normal during the twenty-four hours preceding the establishment of the diagnosis by roentgenography, and therefore it was assumed that the obstruction in all probability was not complicated by strangulation of the bowel. For that reason, it was decided to withhold operation, at least temporarily, and to await developments in the hope that spontaneous relief of obstruction would occur. One circumstance which militated against prolonged delay, however, was the increasing difficulty in administering fluids by parenteral routes.

Fig. 6.—Four months postoperatively. Upright film, ten minutes after ingestion of small amount of barium. Note normal antrum and first portion of duodenum. Barium already present in jejunum.

On the fifth day following the onset of symptoms, the temperature rose to 105°. By this time, fluids no longer could be administered intravenously since no patent veins could be found even by cutting down. At the same time, clyses were being absorbed poorly and the child was losing weight. Vomiting, obstipation, and abdominal distention continued, and, in view of the inability to overcome dehydration, the decision was made to operate. Accordingly, the child was taken to the operating room and, everything being in readiness for rapid exploration, drop-

ether anesthesia was begun. As the anesthetic was being administered, the child began to struggle violently and moved its bowels on the operating table. Anesthesia therefore was discontinued at once.

Feedings were begun within a few hours and were retained. The next day the bowels moved spontaneously and continued to do so regularly thereafter. During the next ten days, the child suffered an episode of severe stomatitis and pharyngitis associated with high irregular fever, which on one occasion reached 108°. At that time convulsions and Cheyne-Stokes respiration occurred but subsided under intensive treatment. Following this episode the course was one of improvement, the temperature slowly subsiding and the local pharyngeal manifestations clearing progressively. At the time of discharge from the hospital, five and one-half weeks after operation and two and one-half weeks after the spontaneous relief of the secondary intestinal obstruction, the wound was firmly healed, bowels were regular, the child was eating normally, and she had regained all of the weight previously lost. Thereafter she was observed regularly by one of us (R. M. S.).

At first the bowels moved four to five times daily, apparently as the result of rapid emptying of the stomach following pyloroplasty, but this symptom was controlled without difficulty by dietary regulation. Follow-up gastrointestinal studies were made four months after operation. A film, taken with the patient in the erect position ten minutes after the ingestion of a small amount of barium (Fig. 6), revealed the antrum and the first portion of the duodenum to be normal in size, shape, and position. A small amount of barium already was present in the jejunum. A film, taken in the prone position thirty minutes after the child began to ingest a full barium meal, revealed the stomach to be well filled. The antrum and the first, second, and third portions of the duodenum were clearly visualized and appeared normal. The head of the column of barium was in the region of the upper ileum, indicating rapid evacuation of the stomach and intestinal hypermotility as the result of pyloroplasty. Subsequent films revealed barium in the large intestine and close inspection failed to reveal the presence of any abnormality of the intestinal canal to indicate the site at which the postoperative intestinal obstruction previously had been present.

The child's present weight, approximately seventeen months after operation, is 30 pounds and she is entirely free of gastrointestinal symptoms. Although her mental and physical development have been normal, the facies strongly suggest the possibility of mongolism.

SUMMARY

1. Congenital obstructions of the stomach, exclusive of hypertrophic pyloric stenosis, are described and their extreme rarity discussed.

2. A unique case of congenital gastric obstruction, due to a complete prepyloric septum of mucous membrane, is described in detail. The patient was a premature infant.

3. Three and possibly all four offspring of the patient's father and the father's two sisters suffered from some congenital anomaly; exclusive of the patient, all died in infancy.

4. The presenting symptoms commenced at birth and were those of high obstruction. Additional clinical features were the failure of the child to pass meconium and the presence of icterus which was more intense than normal. These manifestations suggested the presence of additional obstructive anomalies, of the lower intestinal tract and biliary duct system respectively. X-ray examination indicated the presence of an obstructive lesion in the distal portion of the stomach or very first portion of the duodenum.

5. Operation, performed on the fifth day of life, consisted of multiple incisions of the prepyloric septum and pyloroplasty. No other congenital anomalies were found.

6. The early postoperative course was uneventful. The icterus was concluded to be an accompaniment of prematurity. The failure to pass meconium was assumed to be due to a lack of the normal stimulus to intestinal peristalsis which apparently is initiated by the passage of gastric contents into the duodenum.

7. On the eighteenth postoperative day, the patient developed a secondary, complete intestinal obstruction. Five days later, as operation was about to be performed, the obstruction was relieved spontaneously. The course thereafter, although temporarily stormy, was one of improvement ending in recovery.

8. Follow-up x-ray studies, performed five months after operation, revealed the gastrointestinal tract to be normal except for the presence of rapid emptying of the stomach and intestinal hypermotility, apparently due to the pyloroplasty. The patient more than doubled her lowest weight during that period. The follow-up period now is seventeen months.

9. To the best of our knowledge, this case is the first of its kind to be reported. It also represents one of the very few successful attempts at major surgery upon a premature child and the earliest pyloroplasty. Finally, it appears to be the earliest case of postoperative intestinal obstruction recorded in the literature.

We express our appreciation to all who assisted in the care of the patient, and particularly to the members of the Attending Pediatric Staff and Pediatric House Staff, without whose valuable cooperation we doubt whether the child would have survived.

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CEREBRAL HERNIATION THROUGH THE INCISURA TENTORII*

A Clinical, Pathological, and Experimental Study

William Lister Reid, M.B., M.Sc., Montreal, Quebec

(From the Department of Neurology and Neurosurgery, McGill University, and the Montreal Neurological Institute)

THE openings which connect the anatomical subdivisions of the cranial cavity with each other and with the spinal canal frequently play an important role in the development of certain complications in cases of increased intracranial pressure. These openings are three in number: (1) the foramen magnum, (2) the opening beneath the falx, (3) the incisura tentorii. The boundaries of each are relatively rigid and nonyielding, and when the pressure in one compartment is increased, as by an expanding lesion, the contents tend to be forced through the connecting opening into the adjoining compartment. The signs that may be exhibited under these circumstances depend on the degree of pressure exerted by the herniated tissue on the neighboring structures.

The best known of the herniations occurs through the foramen magnum, particularly in expanding lesions of the posterior fossa. The opening beneath the falx, however, is probably the most frequent site for cerebral herniation and is usually the first opening through which it occurs in supratentorial expanding lesions. This can be seen by the midline shift in the x-ray films after oxygen has been injected into the ventricles.

In this report special consideration is given only to herniation through the incisura tentorii. This opening is triangular in shape (Fig. 1). Anteriorly it is limited by the basilar portion of the occipital bone, and laterally by the rigid free borders of the tentorium cerebelli. On each side immediately above the tentorial edge lies the hippocampal gyrus in such a position that, when pressure is exerted from one of the hemispheres, this structure is the one that is forced over the edge. Within the confines of the incisura, and in close relationship to the hippocampal gyrus, lie the third nerves, the cerebral peduncles, the midbrain, and the posterior cerebral arteries and their branches (Fig. 2). As the free space in the incisura is very limited, it is easily understood how any herniation from above may press on the underlying structures or interfere with the flow of blood through the vessels and produce one or more of the clinical symptoms or pathological changes

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to be described. Herniation through the incisura tentorii is a common occurrence as a late complication in expanding supratentorial lesions. It is my aim to demonstrate the pathological lesions which the herniations cause and describe the clinical signs that such patients may show.

For the purpose of this study the records of 32 patients with supratentorial expanding lesions showing the clinical signs or pathological changes to be described have been studied. Twenty-five of these were examined at necropsy. A variety of supratentorial expanding lesions are included in the series. There were 21 cases with neoplasms, 6 extradural and 2 subdural hemorrhages, 2 intracerebral abscesses and

Fig. 1.—Photograph of base of normal skull with part of right temporal and occipital lobes left in place. *H*, Hippocampal gyrus; *N*, third nerve nucleus; *MB*, midbrain; *OL*, occipital lobe; T_1, free edge of tentorium cerebelli; T_2, attached edge of tentorium cerebelli; *TL*, temporal lobe; *3*, third nerve.

1 tuberculoma. The results from a group of 10 successful experiments in which supratentorial expanding lesions were produced in 10 macacus rhesus monkeys are included. In each of the 25 cases that were examined at necropsy, and in each of the experimental animals there was a herniation over the free edge of the tentorium cerebelli and histologically this herniation always revealed the structure of a distorted, sometimes strangulated, hippocampal gyrus (Fig. 3). In the more marked cases this herniation was projected downward in the form of

a wedge between the tentorial edge and the midbrain. In most instances the herniation was marked, but in some, often contrary to expectation, it was only slight. In the latter cases it was assumed that after death the herniated tissue had shrunken and largely returned to its normal position. The degree of herniation found in the animals was usually much greater than in the clinical cases, because the animal brains were always hardened in situ with formalin to prevent any recession of the herniation. In general, in both clinical and experi-

Fig. 2.—Photograph of base of brain showing the relationship between the herniated hippocampal gyrus, the third nerve, the blood vessels, and the midbrain. *H*, Herniated hippocampal gyrus; *PC*, posterior cerebral artery; *SC*, superior cerebellar artery; *G*, groove in herniated tissue made by the third nerve; *X*, groove made by free edge of tentorium cerebelli; *2*, optic nerve; *3*, oculomotor nerve.

Fig. 3.—Section through the herniation stained with thionin, showing that the herniated tissue has the same structure as the hippocampal gyrus. H_1, Hippocampal gyrus; H_2, herniated tissue. The arrows indicate the position of the tentorium cerebelli.

mental cases, the degree of herniation was roughly proportional to the amount of displacement produced by the primary expanding lesion.

In 17 of the 25 cases that came to necropsy, there was one or more of the following clinical signs present which it seems clear were due to the herniation: (1) ipsilateral third nerve palsy, partial or complete; (2) ipsilateral hemiplegia; (3) evidence of midbrain involvement and, from the pathological standpoint, in addition to the herniation: (a) infarction of one or more occipital lobes; (b) hemorrhages into the midbrain and pons; (c) partial occlusion of the cerebral aqueduct. In the other 8 necropsy cases these features were not observed although there were varying degrees of herniation present. This shows that the herniation may exist without causing any of the above clinical signs and without producing secondary damage to adjacent structures.

Fig. 4.—Showing the apparatus used in the acute experiments and the fixed dilatation of the ipsilateral pupil.

In the experimental animals an attempt was made to reproduce the signs found in human beings by means of an artificial extradural expanding lesion. Under general anesthesia a burr hole was made through the skull, the exact location varying slightly in each animal, but most often situated in the anterior two-thirds of the head. In the acute experiments (Fig. 4), after stripping the dura for a short distance around the burr hole, a Forbes window was screwed into the bone. One outlet was closed off and the other led by a rubber tubing to a large bottle of Ringer's solution. This was connected to a Woulfe bottle, which in turn was connected to the air pressure tap, and a mercury manometer. The fourth outlet from the Woulfe bottle was used as a safety valve. In this manner it was possible to force Ringer's solution into the extradural space at any desired pressure. It was

found that the best pressure to produce the desired results was 90 to 110 mm. of mercury. If greater pressure was used, the animal became deeply comatose and stopped breathing, and would die if the pressure were not released for a few minutes.

In the more prolonged experiments moistened saraka seeds or tragacanth was packed into the extradural space and the burr hole closed by means of a metal screw. The animal was then allowed to recover from the anesthetic and was carefully watched until it died. After death the arterial and venous systems were washed clean with Ringer's solution, and then irrigated with 10 per cent formalin to fix the brain in situ before it was removed. The acute experiments lasted from four to eight hours, while the time for the more prolonged experiments varied from twenty-five hours to four days. The complications resulting from herniation of the hippocampal gyrus over the free edge of the tentorium cerebelli are considered under two headings, first, the clinical signs which it may produce, and second, the pathological alterations it may cause.

CLINICAL SYMPTOMS

Ipsilateral Third Nerve Palsy.—The third nerve carries fibers that supply three separate groups of muscles: (1) the pupilloconstrictor muscle of the iris; (2) the levator palpebrae; (3) the rectus superior, rectus inferior, rectus internus, and obliquus inferior. There is fairly strong evidence to show that the fibers to the iris are separate and distinct from those supplying the other two groups. Hoessly (1918)[1] stated that "the iris fibers were more sensitive than those supplying the extraocular muscles, for they may show paresis without any affection of the extraocular muscles." In our clinical cases and animal experiments this was borne out. In each case in which the changes were closely observed throughout the various stages the pupil first began to dilate and soon lost its reaction to light. This was closely followed by ptosis of the upper lid and then by limitation of inward rotation of the eyeball. In Case 1 this series of events was well demonstrated.

CASE 1.—*Neoplasm of right parietal region. Ipsilateral third nerve palsy.*

A. C., male, aged 10 years. This child was admitted to the hospital complaining of headache, vomiting, drowsiness, and left-sided weakness. He had had transient periods of unconsciousness during which there was generalized rigidity. A biopsy from the right parietal region disclosed a rapidly growing glioma and no further operative interference was considered to be warranted. Before his discharge from the hospital there were three opportunities to observe the attacks of unconsciousness and rigidity inadequately described by the parents as they had seen them at home. In each one of the three episodes the course of events was identical. Generalized rigidity made its appearance and consciousness was rapidly lost. The ipsilateral pupil began to dilate slowly and soon lost its reaction to light. Before the pupil became fully dilated, the upper lid began to droop and it was

then noted that the eye turned out. Though the unconsciousness and rigidity lasted but two minutes, it took about five minutes for the paralytic third nerve signs to disappear.

The patient was discharged from the hospital and, when he died at home, the referring physician was unable to arrange for a post-mortem examination.

In the animal experiments this order of events was reproduced. Case 1 also serves to emphasize the importance of careful and repeated observation of the pupils, for, if they had not been closely watched, the third nerve changes might well have been missed.

It must not be taken for granted that pupillary changes always indicate an intracranial lesion. A dilated pupil, with or without fixation to light, may be due to one of many causes. Although Brooks (1921)[2] believed that unequal pupils were always pathological, Barrie (1918),[3] after examining the eyes of 326 men, found that approximately 10 per cent of them had unequal pupils without any other signs of disease of the eyes or central nervous system.

Local eye disease, such as blindness, glaucoma, optic atrophy, etc., may cause unequal pupils, and certain drugs, such as atropine, homatropine, cocaine, etc., if introduced into the eye, will produce a dilated and fixed pupil. MacDonald (1921)[4] described such a case in which belladonna was accidentally introduced into the eye with a resulting dilated and fixed pupil.

An excitatory lesion in the path of the cervical sympathetic must be considered. Hopkins (1925)[5] observed that unilateral dilatation and fixation of the pupil were sometimes seen in cases of pulmonary tuberculosis, and he assumed that it was due to stimulation of the sympathetic chain by the pressure of the enlarged bronchial glands. He also cited a case in which the pupils were slightly unequal in a patient with syphilis. So it is clear that all extrinsic causes of pupillary changes must be eliminated before assuming that the lesion lies within the cranial cavity.

A detailed account of the evidence that was accumulated with regard to third nerve palsy has already been reported by Reid and Cone.[6] Their conclusions were that ipsilateral third nerve palsy, partial or complete, occurring in cases of intracranial expanding lesions, was due to direct pressure on the third nerve by a herniation of the hippocampal gyrus over the free edge of the tentorium cerebelli. This is well demonstrated in Fig. 2 where the third nerve is flattened and has formed a groove in the herniated tissue.

In some of the clinical and experimental cases that have been studied with regard to this feature, one or more of the other five features were also present, but in each case the signs of third nerve palsy were observed first. In other words, when herniation of the hippocampal gyrus occurs, the ipsilateral third nerve, being the closest structure, is the first to feel the effects of pressure exerted by the herniated tissue. As the herniation increases, it may press on one or more of the

other structures in the incisura with the production of signs depending on the structure that is involved.

Ipsilateral Hemiplegia.—Not infrequently cases are seen in which there is an ipsilateral hemiparesis or hemiplegia associated with an intracranial expanding lesion. This may be confusing as a localizing sign, and it has often led to an exploratory operation on the wrong side. With modern methods of visualizing the ventricular system the possibilities of such an error have been greatly diminished. Even now, however, in certain cases of head injury the surgeon usually makes a burr hole on each side of the skull, partly to avoid the chance of an error in localization and partly because of the possibility of a bilateral hemorrhage.

Purves-Stewart (1927),[7] Flateau (1924),[8] Putnam and Cushing (1925),[9] Groenveld and Schaltenbrand (1927),[10] Woltman (1928),[11] and others have reported such cases of ipsilateral hemiplegia. In each of the seven cases described by Rand (1929)[12] in relation to ipsilateral dilatation of the pupil in cases of intracranial hemorrhage, there was also ipsilateral hemiplegia. Kernohan and Woltman (1929)[13] published a very concise report of 42 cases of intracranial expanding lesions, 37 of which were supratentorial. The reason for this report was to explain the mechanism by which ipsilateral hemiplegia was produced in such cases. Their conclusion was that it was caused by compression of the opposite crus cerebri against the sharp rigid free edge of the tentorium cerebelli, producing a notch in the crus and destruction of the tissue at the point of contact. They also observed that there may be herniation through the incisura tentorii as evidenced by the groove along the uncinate gyrus on the side of the primary expanding lesion. Twenty-two of their cases showed ipsilateral hemiplegia and notching of the crus; 2 showed mild ipsilateral signs without any notching; and the remaining 18 showed notching of the opposite crus but no signs of hemiplegia.

Hoen (1935)[14] described 3 cases of cerebral expanding lesions with signs of ipsilateral weakness or paralysis. He, too, believed that it was due to the pressure of the lesion pushing the brain toward the opposite side and compressing the midbrain against the tentorial edge. He felt that the syndrome produced was diagnostic of a lesion in the frontoparietal region.

In our own experimental studies 2 of the animals developed ipsilateral hemiplegia. In each case this was preceded by ipsilateral fixed dilatation of the pupil and ptosis of the upper lid. At necropsy marked herniation of the hippocampal gyrus over the free edge of the tentorium was found, but there was no notching of the crus, nor were any pathological changes found in the midbrain microscopically. In only 1 of the 32 clinical cases (Case 2) was there ipsilateral hemiplegia.

CASE 2.—*Large extradural hematoma left temporoparieto-occipital region. Ipsilateral third nerve palsy, hemiplegia, and occipital lobe infarction.*

S. W., male, aged 30 years. This patient was unconscious for some time following a head injury. After a lucid interval of an hour or so he again became uncon-

scious and the attending physician noted a dilated and fixed pupil and hemiplegia on the left side. On admission to hospital he was in deep coma and both pupils were dilated and fixed.

At operation a large hematoma was found on the left side extending from the temporal region well back over the occipital lobe. Although the brain returned to its normal position and was pulsating well after the removal of the clot, the patient's condition did not improve and he died four days later.

At necropsy the following points were noted: (1) herniation of the left hippocampal gyrus over the tentorial edge; (2) slight indentation of the right crus cerebri with hemorrhagic softening of its substance (Figs. 5 and 6); (3) large area of infarction in the left occipital lobe and a small one in the right.

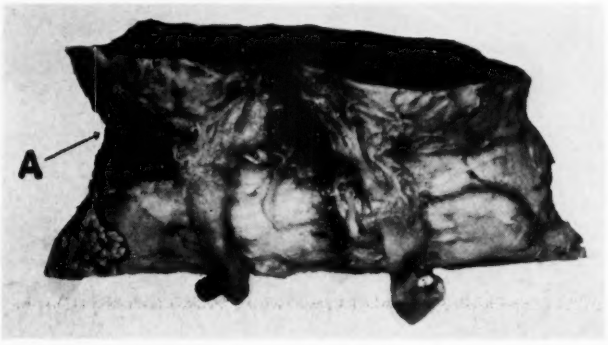

Fig. 5.—Showing notch in right crus cerebri made by the tentorial edge; *A*, notch.

Fig. 6.—Showing hermorrhagic softening of the right crus cerebri. *A*, Right crus; *B*, infarction in left temporal and occipital lobes.

From the work of Kernohan and Woltman the mechanism involved in this feature is quite clear. As they have shown, if the primary lesion is large enough it pushes the brain toward the opposite side and forces the opposite crus against the sharp free edge of the tentorium, causing

an interference with the conduction of nerve impulses and resulting in weakness or paralysis on the ipsilateral side. In addition to this I wish to emphasize the relationship that exists between the hemiplegia and the herniation of the hippocampal gyrus. It is my belief that, when the pressure exerted by the primary expanding lesion is sufficient to force the midbrain against the opposite tentorial edge, herniation of the hippocampal gyrus is invariably present. I also feel that the herniation is important in providing an additional and more direct line of force in pushing the midbrain against the opposite tentorial edge.

Signs of Midbrain Involvement.—One often sees a patient with a supratentorial expanding lesion admitted to the hospital with signs of midbrain involvement, such as deep coma, spasms of rigidity like those of a decerebrate animal and set off by the slightest stimulus, hyperpnea, tachycardia, and hyperthermia. These symptoms may be due to the pressure of the herniated hippocampal gyrus on the midbrain or to hemorrhage into the midbrain as will be pointed out later on.

When the pressure of the primary expanding lesion is great enough to force the hippocampal gyrus downward over the tentorial edge and through the incisura tentorii, the herniated tissue acts as a wedge inserting itself between the free edge of the tentorium laterally and the midbrain medially. The midbrain is thus compressed between the herniated tissue on the one side and the opposite tentorial edge on the other. The effects of the pressure exerted by these two structures differ in many respects. As the midbrain is forced against the sharp rigid tentorial edge, the damage that is done is mostly confined to the point of contact which, in this case, is the crus cerebri. As has been shown by Kernohan and Woltman, this results in an ipsilateral weakness or paralysis. On the other hand, the herniated tissue is broader, softer, and more pliable, so that the effect of its pressure is spread more evenly throughout the midbrain. The latter pressure is probably the main factor in the production of the signs of coma, decerebrate rigidity, etc., signs that indicate a near terminal or terminal stage in these cases.

Further evidence in this regard was obtained from two of the experimental animals. In each case fluid pressure was used forming an extradural hygroma which was connected to the air pressure tap and a mercury manometer. When the pressure was raised rapidly to 110 mm. of mercury, each animal became unconscious, the ipsilateral pupil dilated and soon lost its reaction to light, the upper eyelid dropped, and inward rotation of the eyeball was lost. There was marked spasm resembling that seen in decerebrate rigidity; respiration became labored and then ceased, and the heart beat stopped. All this took place in about ten seconds. On release of the pressure the animals rapidly recovered. This procedure was repeated several times in each animal with the same result. Finally, the pressure was maintained at 110 mm. of mercury until the animals died and the brain was then fixed in situ. At

necropsy, in each case, there was seen marked herniation of the hippocampal gyrus forming a wedge between the free edge of the tentorium and the midbrain (Fig. 7). A similar series of events was seen in Case 3.

CASE 3.—*Large intracerebral neoplasm in right temporoparietal region.*

W. S., male, aged 45 years. Prior to operation a ventriculogram was done and the patient placed on the x-ray table. While the pictures were being taken, he suddenly became rigid and stopped breathing and it was noted that the ipsilateral pupil had become dilated and fixed to light. A brain needle was immediately inserted into the ventricle and some fluid and gas escaped under fairly high pressure. The patient recovered rapidly to his original condition and the pupils returned to normal more slowly.

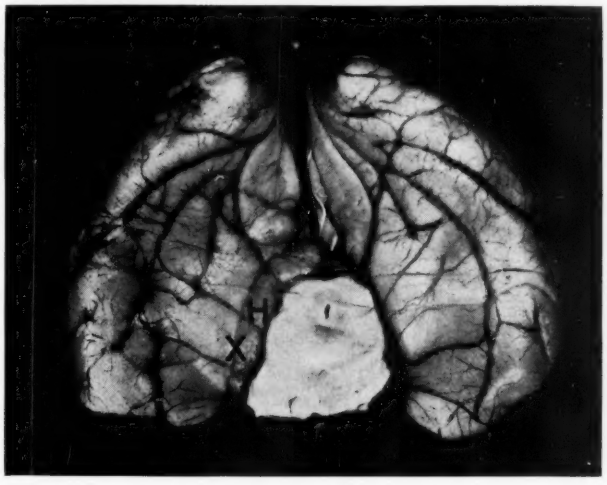

Fig. 7.—Experimental animal with extradural hygroma, showing herniation of the hippocampal gyrus forming a wedge between tentorium and midbrain and compression of the latter. Also showing narrowing of cerebral aqueduct. *H*, Herniated hippocampal gyrus; *X*, groove made by the sharp tentorial edge.

When considering the mechanism of pressure on the midbrain in cases of supratentorial expanding lesions, it is clear that the herniated hippocampal gyrus is of the utmost importance and it may be necessary to treat the herniation as well as the primary expanding lesion. Jefferson (1938)[15] reported one case with a right temporal lobe neoplasm and an ipsilateral dilated and fixed pupil. He removed the entire temporal lobe together with the herniated tissue and the patient made a complete recovery. Vincent, David and Thiébault (1936)[16] stated that they had removed the herniated tissue in certain cases and that the patients had recovered. In two of my own cases the herniations were not relieved by the removal of the extradural hematomas which had produced them and the patients died. It seemed from the post-mortem examinations that the herniations must have receded somewhat after the removal of the primary lesion but that the pressure on the midbrain was not completely relieved. Therefore, in agreement with Jefferson, it is suggested that the surgical procedure must also include

either manipulations to prove the hernia is reduced or removal of the herniated tissue if reduction is not complete. It may in some instances be advisable to enlarge the opening by incision of the free edge of the tentorium. The fact that in some of our recovered cases this was not done shows the herniation may correct itself, but it is leaving less to chance if the region is at least inspected, the necessary retraction for exposure of the area being itself of value to reduce a small hernia after the primary cause of the intracranial pressure increase has been corrected.

PATHOLOGICAL CHANGES

Infarction of One or Both Occipital Lobes.—This is a comparatively rare complication but one that comes within the scope of this study. Meyer (1920),[17] in discussing herniation of the brain, described several cases of supratentorial expanding lesions that showed a "tentorial line" along the uncus where it had herniated over the edge of the tentorium cerebelli. In one of these cases the blood flow through the right posterior cerebral artery was interfered with by the herniation, and this resulted in collapse of the corresponding part of the occipital cortex. Another case in the same series revealed a much larger herniation, but there was no evidence of interference with the blood flow. Moore and Stern (1938)[18] described five cases in each of which there was occipital lobe infarction associated with an intracranial expanding lesion. They felt that the infarctions were due to compression of the posterior cerebral artery.

In four of my own series of cases there was occipital lobe infarction. In each case there was a unilateral intracranial expanding lesion which was associated with ipsilateral fixed dilatation of the pupil and herniation of the hippocampal gyrus over the free edge of the tentorium cerebelli. In three of these cases the occipital lobe infarction was bilateral but more marked on the ipsilateral side. In each of these 3 cases there was bilateral herniation of the hippocampal gyrus, but also more marked on the side of the primary expanding lesion. In the fourth case the infarction was unilateral and on the same side as the expanding lesion, as also were the herniation and the third nerve palsy.

At necropsy we were unable to make out the exact mechanism involved in interfering with the blood supply to the occipital lobes, but it was evident that the herniated hippocampal gyrus was an essential part of that mechanism. Because of the rarity of this condition as compared with the frequent occurrence of the herniation, and the fact, as seen in Meyer's two cases, that infarction is not necessarily dependent on the size of the herniation, we feel that the interference with the blood supply to the occipital lobes is probably due to some anomaly in the anatomical course of the vessels. In this manner the arterial obstruction is due to stretching or kinking of the vessel by the herniation rather than to direct compression.

In our animal experiments the extradural pressure necessary to produce even marked herniation was always less than the systolic blood pressure. The pressure at the point of the herniation would be less still, and probably much less than would be necessary to compress the artery directly. It is possible that, once the herniated wedge is in position, the addition of edema in the herniated tissue may supply the additional pressure necessary to compress the vessel, but, if this were the case, one would expect occipital lobe infarction to be much more common.

The points that we wish to emphasize in relation to occipital lobe infarction occurring in cases of supratentorial expanding lesions are: first, the constant presence of herniation of the hippocampal gyrus over the free edge of the tentorium; second, that this herniation is an essential part of the mechanism involved in the production of the infarction; and, third, the constant association of ipsilateral third nerve palsy in each of these cases.

Hemorrhage Into the Midbrain and Pons.—It is not at all uncommon, on sectioning the brains of patients who have died from an intracranial expanding lesion, to find hemorrhages in the midbrain and pons. These hemorrhages vary considerably in size. Some consist merely of a diapedesis of blood cells around the vessels, while in others there may be massive hemorrhages with considerable destruction of the surrounding tissue. Moore and Stern reported eleven such cases and they believed that the herniated hippocampal gyrus was an essential part of the process involved. Our own studies have helped to confirm this viewpoint, but the evidence that we have obtained has not been sufficient to enable us to formulate any theory as to the exact manner in which the herniation produces the hemorrhage.

In 9 of the 25 brains of this group that were examined at necropsy, there were midbrain and pontine hemorrhages. In 7 of these 9 cases signs of third nerve palsy were observed before death and in 1 of this group there was bilateral occipital lobe infarction. Also in all 9 cases there was herniation of the ipsilateral gyrus over the free edge of the tentorium. Observations add nothing to and only confirm the findings of Moore and Stern as far as the midbrain hemorrhages are concerned, but I do want to emphasize that, when herniation caused midbrain and pontine hemorrhage, the presence of the herniation was at least suggested before death in 7 of the 9 cases by the ipsilateral third nerve palsy.

Obstruction of the Aqueduct of Sylvius.—In many of the cases examined at necropsy the cerebral aqueduct has been so greatly distorted and displaced by the herniation that it would seem to have been functionally blocked. This feature has been more clearly demonstrated in some of the experimental animals where the brains were fixed in situ by intravascular injection before removal. In Fig. 7 the aqueduct is seen to be narrowed. In another monkey (Fig. 8) it was almost completely occluded.

We have only cursory clinical evidence to show that the aqueduct may be obstructed by herniation since it has not been our custom to take simultaneous ventricular and lumbar pressures. The work of Smyth and Henderson[19] proves the point conclusively. Eight of 33 tumor cases which they studied showed a disturbance of dynamics. Six of these had supratentorial tumors and they had an initial pressure difference, the ventricular readings being from 20 to 100 mm. of water higher than the spinal ones. This disparity was accentuated by the withdrawal of lumbar fluid. In these 6 cases jugular compression also proved a partial block. When the ventricular pressure was higher than

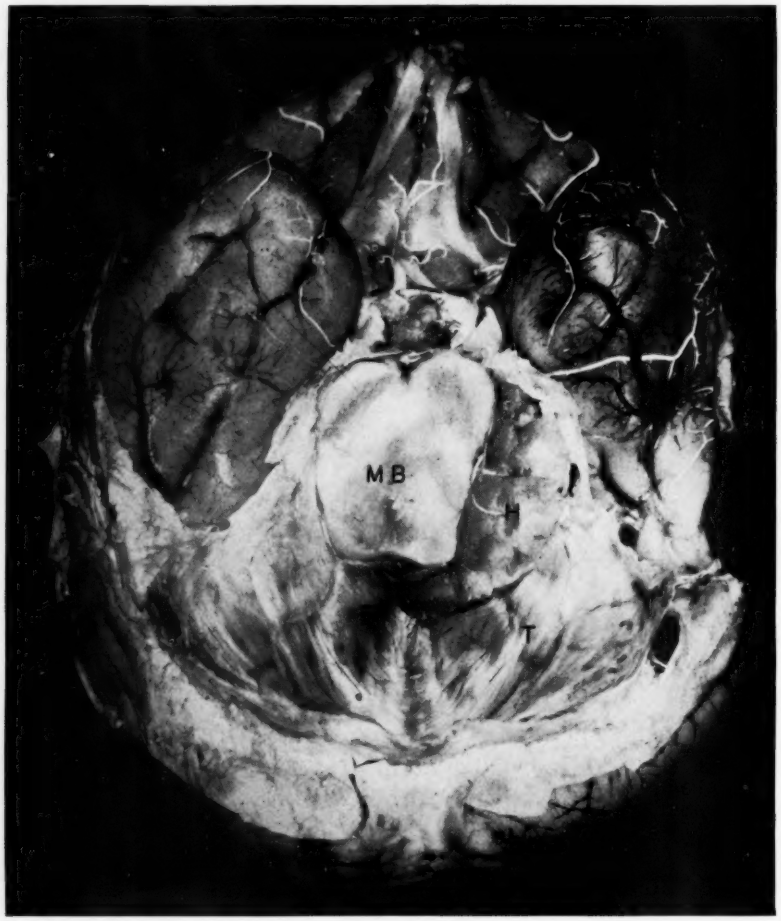

Fig. 8.—Showing a large herniation of the hippocampal gyrus with compression of the midbrain and almost complete occlusion of the cerebral aqueduct. *MB*, Midbrain; *H*, herniation.

the lumbar pressure there was a high operative mortality. The six patients died and post-mortem evidence of herniation of the temporal lobe through the incisura tentorii was found.

I question only one of the statements in their discussion, though the points in my experience leading me to query it are not beyond criticism. They state: "It is apparent that the observations here recorded afford

conclusive proof that the lumbar and ventricular pressures are not of necessity always equal.'' I concur in this, but, when they add, ''a difference was observed only in cases where the lumbar pressure was abnormally high,'' and then go on to say, ''it seems therefore that a normal lumbar puncture pressure in a case of intracranial tumor is a true index of the intracranial pressure,'' I take issue. While it holds in the cases they studied, it seems to me to be unwarranted as a general conclusion as the following case suggests.

CASE 4.—B. O., female, aged 25 years, had suffered right frontal head injury one-half hour before admission to the hospital. She had recovered consciousness after a few minutes and then was nauseated and her senses became dull. The left abdominal reflexes were absent and the left biceps and knee jerk were slightly accentuated. The pulse was 100 and the blood pressure was 128/72. The spinal pressure was 120 mm. of water and the spinal fluid was bloody. Following the lumbar puncture her senses became more dulled; left-sided weakness with accentuated deep reflexes developed. The pulse fell to 76 and then to 66. A second lumbar puncture was done and the pressure was 60. The right pupil dilated and became fixed to light; respirations became stertorous and the patient became deeply comatose. Following hypertonic glucose the general condition improved slightly. Lumbar puncture then showed the pressure to be too low to record.

Because of the low spinal pressures it at first was decided that an extradural hemorrhage could not be present and that the symptoms were due to advancing hemorrhages in the midbrain. As the patient's condition became worse a subtemporal exploration was carried out four hours after the accident in spite of the low spinal pressure because the course of events was so typical of extradural hemorrhage. An extradural clot and fluid blood with an estimated volume of 300 c.c. was present, producing much pressure and much deformity. The bleeding point from the middle meningeal artery was easily secured. The brain expanded but little after the clot was removed. She died two days later never having recovered consciousness.

It was the 1931 notes of this catastrophe that led us to study extradural compression experimentally and led on to an investigation of the temporal pressure cone. The case illustrated the point not only that lumbar puncture in such cases may be followed by a marked progression of signs but that lumbar pressures may give no true index of the intracranial pressure.

SUMMARY

As supratentorial intracranial expanding lesions increase in size, pathological alterations may take place at a distance from the primary lesion due to brain shift and herniation into adjacent intracranial compartments. The extent of the herniation and shift depends on the force exerted by the primary lesion and the clinical signs depend on the structures encroached on by the displaced brain.

Herniation of the hippocampal gyrus of the temporal lobe through the incisura of the tentorium cerebelli is a common occurrence in supratentorial space-occupying lesions. Clinically it can cause ipsilateral third nerve paresis or paralysis, ipsilateral hemiplegia, signs of midbrain involvement, and obstruction to the aqueduct of Sylvius. Grave irreversible damage to important structures may be produced

by it, such as infarction of the occipital lobes and pontine hemorrhages, damage to the crura cerebri and hemorrhage and softening of the herniated gyrus.

As has been shown in a previous report, one of the earliest signs of incisural herniation that will cause damage is dilatation of the ipsilateral pupil. Dilatation of the ipsilateral pupil has commonly occurred in this series of patients before ipsilateral paralysis of the extremities and the other signs of graver damage. It is important to recognize and evaluate properly the significance of the secondary clinical signs that occur as a result of these herniations and realize the pathological alterations that result from them if prompt treatment is not instituted. The herniation of the hippocampal gyrus may not recede after the primary expanding lesion is removed and the surgeon must keep this possibility in mind, for Jefferson and others have removed the strangulated gyrus after treating the primary lesion with recovery of the patient.

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CEREBELLAR SUBDURAL HEMATOMA IN INFANT 2 WEEKS OLD WITH SECONDARY HYDROCEPHALUS

Operation With Recovery

Richard G. Coblentz, M.D., Baltimore, Md.

(From the Departments of Pathology and Neurosurgery, University of Maryland Medical School)

THE formation of a subdural hematoma following intracranial hemorrhage in the newborn probably occurs more frequently than is generally recognized. In fact, Ingraham and Heyl[1] in a recent article state that "it is a common condition in infancy and early childhood" and that "the frequency with which subdural hematoma is found among children is more or less proportional to the intensity with which it is sought." This statement is, perhaps, particularly true in young infants, in whom a subdural clot is more apt to be accompanied by subarachnoid bleeding which may mask the presence of the subdural lesion. Failure to recognize either of the two types of hemorrhage, if the infant survives for any length of time, frequently results in the development of a secondary hydrocephalus due to (1) interference with the absorption of the cerebrospinal fluid or (2) the mechanical obstruction to flow of the cerebrospinal fluid from the ventricular system.

It is a well-recognized fact that trauma is, perhaps, the most frequent cause of subdural hematoma in adults, but that systemic diseases, especially those affecting the vascular system of the brain, may play an important secondary role. In infants and children trauma is considered an important etiologic factor by Ingraham and Heyl.[1] Other investigators (Sherwood,[2] Peet and Kahn,[3] Rosenberg,[4] and Ingalls[5]) call attention to the fact that subdural hematoma occurs frequently in undernourished infants, infants with scurvy and other dietary deficiencies, and particularly in those with vitamin C deficiency in which no history or evidence of trauma can be obtained.

As in adults, the location of the subdural hematoma in infants is almost invariably over one or both of the cerebral hemispheres. Very rarely does an encysted subdural clot form in the posterior fossa over the cerebellar hemispheres, either in adults or infants. Dandy[6] has reported a case in which an encysted clot was found in the pituitary region, producing symptoms of a pituitary tumor. The lesion was found at operation and the patient recovered after evacuation of the clot. Putnam and Cushing[7] cite a case of Naffziger[8] in which the hematoma was chiefly basal in the region about the mastoid, fading out toward the vault. This patient also recovered after operation. The lesion involved the spinal cord also in several cases in the literature.[9]

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In a fairly extensive review of the literature only four cases of encysted subdural hematoma in the posterior fossa over the cerebellum were reported. All these cases were found at autopsy and had not been recognized before death. Neisser and Pollack[10] mentioned two cases found at autopsy, cited by Putnam and Cushing.[7] Picken[11] reported one case found at autopsy, no operation, included in a report of a series of cases by Jelsma.[12] The fourth case, reported by Munro,[13] was a right subdural hematoma, together with a cerebellar subdural hematoma. Death occurred eleven days after evacuation of a right cerebral clot, with the discovery of bilateral cerebellar hematoma at autopsy. In no instance was there a record of an encysted subdural hematoma over the cerebellum which had been recognized and cured by operation.

The chief object of this paper is to record a single case of encysted subdural hematoma over the right cerebellar hemisphere, with secondary hydrocephalus, in an infant 2 weeks old, verified at operation, with recovery.

CASE REPORT

Baby K., aged 9 days, was transferred to the Neurosurgical Service from the Obstetrical Department of the University Hospital on Oct. 24, 1939, because of enlargement of the head and bulging fontanelles, with marked separation of the cranial sutures.

The note on the obstetrical chart was as follows: "Mother admitted from the Out-Patient Department on Oct. 15, 1939, at term and in active labor. Multipara ii. Normal deliveries. Prenatal course with this pregnancy uneventful. Labor progressed normally under routine nembutal and paraldehyde analgesia, and after two hours and forty-three minutes was delivered of full-term, living child, 7 lb. 15¼ oz., by low forceps. Central episiotomy. Third stage uneventful. Cervical inspection negative. Infant shows no anomalies. No cyanosis, convulsions, or snuffles. Moderate occipital caput. Measurements: length, 49 cm.; weight, 7 lb. 15¼ oz. Head: O.M., 13 in.; O.F., 11; S.O.B., 10; B.P., 9; B.T., 8. Treatment eyes, Credé; treatment cord, Credé."

The baby was breast fed with supplementary feedings. He nursed well and appeared and acted normally until the fourth day, at which time the head appeared slightly enlarged and the fontanelles full. During the next four days the head gradually increased in size, the fontanelles were bulging, and there was marked separation of all the cranial sutures. Small areas of ecchymosis appeared in the upper lids of each eye. There was a gradual loss of weight in spite of the fact that the baby retained all [illegible] feedings. On Oct. 24, nine days after birth, the neurosurgical consultation revealed the following: temperature, 98° F.; pulse, 110; respiratory rate, 30. The baby was rather drowsy, but aroused easily and cried normally. The head was definitely enlarged, the greatest circumference being 14½ inches. The fontanelles were tense and bulging. All the cranial sutures were markedly separated. The frontal bosses were not prominent, and there was very little, if any, distention of the veins of the scalp. The upper lid of each eye showed ecchymotic areas at the inner and upper margins of the orbit. There was slight, but definite, cervical rigidity. The pupils were moderately dilated, equal, and reacted to light. The fundi were normal, the vessels full, and there was no choking. There was no facial weakness. The mucous membranes of the mouth

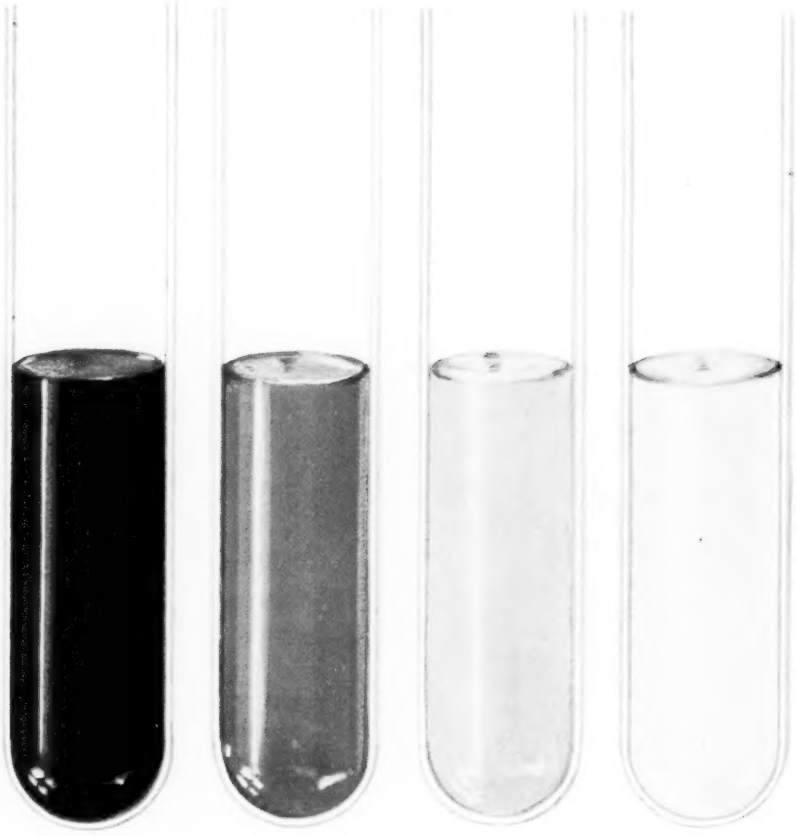

Fig. 1.—Spinal subarachnoid fluid, showing elimination of blood by spinal drainage.

were normal. The deep reflexes present in all four extremities were hyperactive and equal. Plantar stimulation produced dorsal extension at times and dorsal flexion on subsequent examinations. There was no spasticity of the upper or lower extremities. There was no apparent weakness in the arms or legs.

Blood examination: Hb., 114 per cent (Sahli, 16.7 Gm., 14.5 Gm. equaling 100 per cent); R.B.C., 5,340,000; W.B.C., 10,300. Differential: P.M.N., 48 per cent (filamentous 45 per cent, nonfilamentous 3 per cent); lymphocytes, 47 per cent; monocytes, 2 per cent; eosinophiles, 2 per cent; basophiles, 1 per cent. Platelets, 874,500. Bleeding time: 1 minute 45 seconds. Clotting time: 2 minutes. Wassermann negative. Stool normal. Urine negative. Circumference of head: 14½ inches.

X-ray examination of the skull on Oct. 25, 1939, showed marked separation of all the cranial sutures. No other abnormalities were demonstrated. The x-ray examination of the chest showed the lung fields clear, and the diaphragm, heart, and great vessel outlines were normal. Spinal fluid examination: grossly bloody, xanthochromic fluid; 15 c.c. removed.

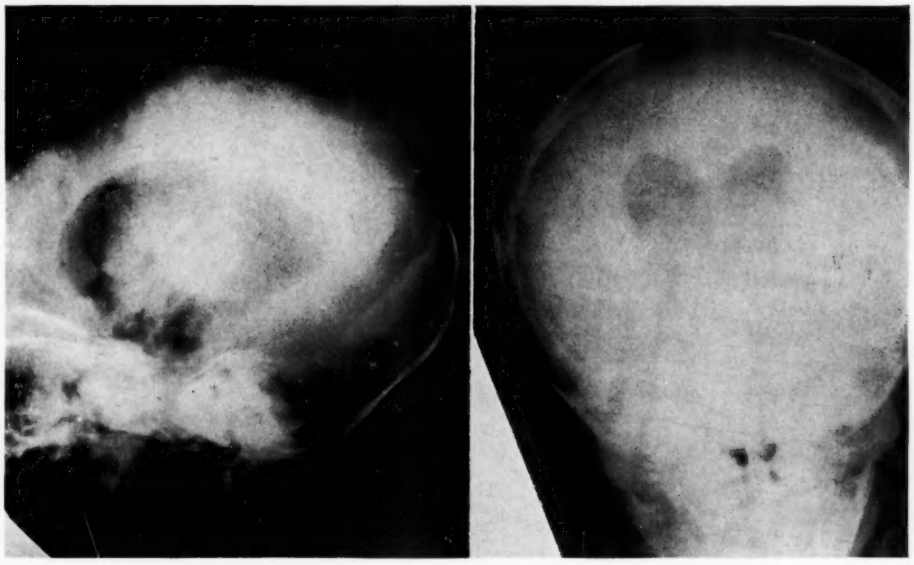

Fig. 2.—X-rays of skull, showing symmetrical dilatation of the ventricles and marked separation of the cranial sutures.

A diagnosis of subarachnoid hemorrhage was made and daily lumbar punctures were done with the following findings: *Oct. 25:* Less bloody xanthochromic fluid; 12 c.c. obtained. Fontanelles soft after puncture. Head: 14½ inches in circumference. *Oct. 26:* Slightly blood-tinged xanthochromic fluid; 10 c.c. obtained. Head: 14¾ inches in circumference. *Oct. 27:* Orange-colored fluid; 8 c.c. obtained. Head: 14¾ inches in circumference. *Oct 28:* Light amber-colored fluid; 4 c.c. obtained. Fontanelles remained distended. Head: 14¾ inches in circumference. *Oct. 29:* Clear, straw-colored fluid; 4 c.c. obtained. Fontanelles tight. After puncture, head was 15 inches in circumference. (Fig. 1.) *Oct. 31:* Bilateral subdural taps through the coronal sutures showed clear fluid on both sides. Bilateral ventricular punctures through coronal sutures yielded slightly xanthochromic fluid. One hundred cubic centimeters of fluid were removed and replaced with air. X-ray examination of the head after air injection showed moderate symmetrical dilatation of the entire ventricular system, with widely separated sutures (Fig. 2). The presence of a subdural hematoma in the posterior fossa over the cerebellum was suspected.

Operation (Nov. 1).—Ether anesthesia; cerebellar position. Through a small semielliptical incision over the occipital region, the occipital bone over the right cerebellar hemisphere was exposed in the usual manner. A small perforation was made in the bone and this opening enlarged to expose the underlying dura, which presented the typical bluish appearance of an underlying clot. The dura was then carefully incised, exposing the outer wall of the sac of a subdural clot. The cyst wall was well formed, the outer wall adherent to the under surface of the dura and grayish in color. When the sac was incised, 25 to 30 c.c. of dark, reddish brown, rusty-looking fluid escaped. No solid clots were seen. Following the evacuation of the clot, close to the midline the arachnoid, forming the posterior wall of the cisterna magna, could be seen bulging to the mesial side of the sac membranes. The bulging arachnoid was pricked with a needle, releasing clear, slightly xanthochromic fluid. The sac was irrigated with saline solution and the incision closed in layers in the usual manner, without drainage. The baby stood the operative procedure well, and immediately following the operation the fontanelles were sunken and there was a marked decrease in the separation of the sutures.

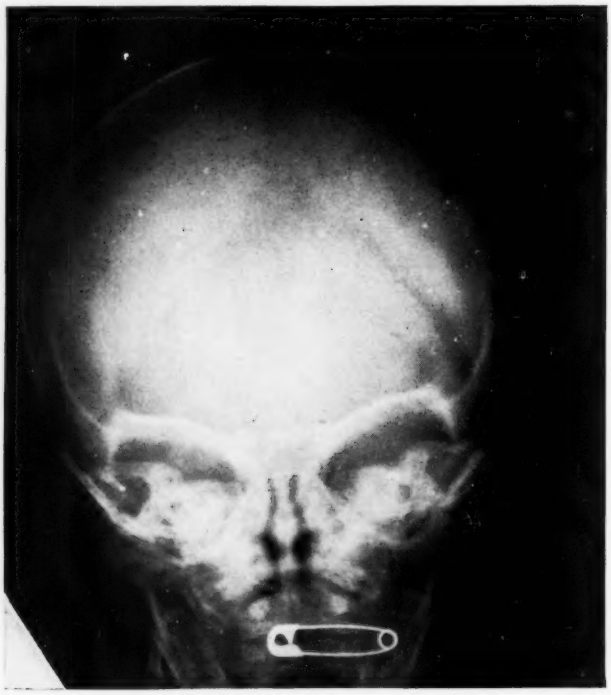

Fig. 3.—X-ray of skull on discharge from the hospital on Nov. 29, 1939, showing normal sutures.

Postoperative Course and Treatment.—The infant was given 50 c.c. each of citrated blood and 0.9 per cent sodium chloride solution immediately after the operation, and this was repeated five and one-half hours later. Ventricular and lumbar punctures were done twelve hours after the operation. Slightly xanthochromic fluid was obtained from both the ventricles and the lumbar subarachnoid space. Infusion of 0.5 ounce of salt solution and 15 c.c. of glucose was given every half-hour for twenty-six hours, beginning two hours after operation. The postoperative course was uneventful. The temperature twelve hours after operation rose to 102° F., but was normal twelve hours later and remained so through-

out the course in the hospital. The fontanelles remained soft and the marked separation of the sutures gradually disappeared. X-ray examination of the skull at the time of discharge from the hospital showed the sutures to be normal (Fig. 3). The baby was discharged from the hospital on Nov. 29, 1939, twenty-eight days after operation, having made a complete recovery (Fig. 4).

Fig. 4.—Photograph of patient three months after operation.

COMMENT

Although there was no definite history of trauma in this case, the ecchymosis of the upper lids and the presence of a moderate occipital caput would lead one to suspect that slight trauma was the most likely etiologic factor. The prompt disappearance of blood in the lumbar subarachnoid fluid following daily lumbar punctures and the continued evidence of increased intracranial pressure indicated a coexisting lesion. Subdural hematoma was ruled out over the cerebral hemispheres by subdural taps through the coronal sutures. Ventriculography definitely located the lesion in the posterior fossa. From this case, I am fully convinced that early investigation of the subdural space and ventriculography are of prime importance in respect to diagnosis and localization. Delay may result in serious damage to the rapidly developing brain, especially when the clot is in the posterior fossa. Evacuation of the clot through a small trephine opening was selected (1) because of the

age of the infant and (2) because we know it is not necessary to remove the entire sac to effect a cure.

CONCLUSIONS

1. Subdural hematoma in the posterior fossa in infants is rare, but can and does occur and should be kept in mind as a possibility in all cases of hemorrhage in the newborn.

2. In infants early bilateral subdural taps through the coronal sutures are justified and indicated to rule out subdural bleeding, especially where there are signs of rapidly increasing intracranial pressure.

3. Ventriculography is a definite aid to localization, especially when the clot is in the posterior fossa.

4. A symmetrical dilatation of the ventricular system develops when the clot obstructs the flow of the cerebrospinal fluid from the ventricles.

5. Evacuation of the clot through a small trephine opening without drainage effected a cure in this case. It is a simple procedure which can be used to verify the diagnosis and to evacuate the clot at the same time.

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NOTE ON STRAIGHTENING THE HYPOSPADIAC PENIS

C. D. Creevy, M.D., Minneapolis, Minn.

(From the Urological Division of the Department of Surgery at the Medical School of the University of Minnesota)

THIS brief paper has been prompted by recurring statements in the literature to the effect that Duplay's plastic operation, devised in 1874, is inadequate for correcting the ventral curvature of the penis associated with hypospadias; such a statement is usually accompanied by a description of a new method of dealing with the penile skin.

Correction of the deformity depends not upon what is done to the skin, but upon complete removal of the fibrous band beneath the corpora cavernosa. This band undoubtedly represents a rudimentary corpus spongiosum urethrae which, by its shortness and attachment to the intact urethra behind, to the corpora cavernosa above, and to the glans penis in front, serves as the bow string which produces the characteristic ventral curvature of the penis.

Duplay excised this fibrous band through a transverse incision between the external meatus and the glans, and closed Buck's fascia and skin longitudinally. Dissatisfaction with the results of this method led Hagner to correct the curvature by implantation of tubular Thiersch grafts transversely in the fibrous band between the corpora cavernosa and the ventral skin; these were later cut through to the ventral surface of the penis. Edmunds devised a two-stage operation in which a transverse buttonhole was made in the foreskin at the first session. Later the fibrous band was excised and the divided foreskin was utilized in a plastic closure. McIndoe has recently stated that this method is often required because of the inadequacy of Duplay's operation. Borchers has recently advocated slipping the penis under a double-ended pedicle graft of abdominal skin just above the symphysis. The ends of this flap are divided later. McKenna has excised the fibrous band and implanted a pedicle graft from the scrotum into the defect on the undersurface of the penis.

The methods of Hagner, Edmunds, and Borchers involve two operations; those of Borchers and McKenna leave hairy skin in the area from which the urethra must be constructed; Cabot has found that the hair may continue to grow and become encrusted with urinary salts. Duplay's method has neither of these disadvantages and, when properly done, achieves results as good as, or better than, any other procedure. There is always plenty of skin on the hypospadiac penis to permit complete straightening if the fibrous band is excised completely, so that the type of skin plastic is of very little moment.

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An additional factor in production of the curvature is the fact (which is rarely mentioned in the literature) that the transition from normal urethra behind to fibrous rudiment in front is often gradual rather than abrupt. That is, in the majority of cases of hypospadias, the terminal portion of the intact urethra has a wall thinner and shorter than normal and so itself contributes to the ventral curvature of the penis.

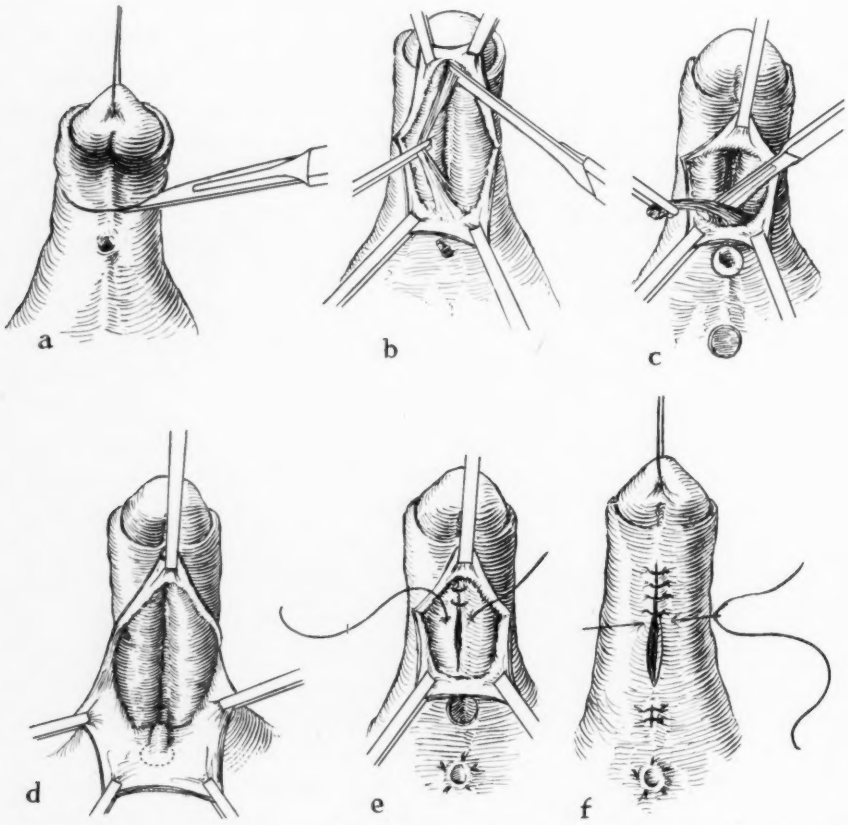

Fig. 1.—Duplay's plastic operation, with the addition of freeing and posterior displacement of the urethra.

a, Incision;
b, freeing of the fibrous band;
c, removal of the band and preparation for posterior displacement of the external meatus;
d, urethra for some distance behind the meatus has been freed from the corpora;
e, posterior displacement of the meatus and closure of the fascia;
f, closure of the skin.

In order to correct this curvature it is therefore essential not only to remove absolutely all of the band from the posterior edge of the glans penis to the external urinary meatus, leaving the tunica albuginea of the corpora cavernosa in this area completely bare, but also, as I have previously pointed out, to free the intact but deficient terminal portion of the urethra from the corpora until the penis can be straightened without traction (Fig. 1). In the more extreme types this may mean separating the urethra entirely from the penis. This has been perfectly

apparent in several of my own cases in which some curvature of the penis remained despite complete excision of the band, but disappeared after separation of the urethra from the corpora cavernosa.

Since Duplay's method of removing the remnant through a transverse incision between the meatus and the glans gives adequate exposure for carrying out this separation, and since longitudinal closure of this incision always leaves plenty of skin on the ventral surface of the penis, there is no need to employ the two-stage operations of Edmunds, Hagner, or Borchers, or the one-stage method of McKenna with its use of hairy skin.

Fig. 2.—A. L. before operation on Sept. 8, 1939.

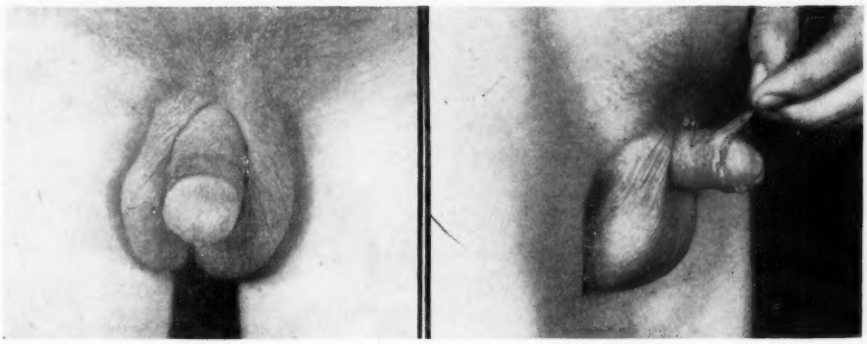

Fig. 3.—A. L. two weeks later.

The result obtained in the most recent of a series of twenty cases of hypospadias will serve to support this contention. The patient, A. L., University Hospital No. 669866, aged 15 years, had the most extreme form of curvature that occurs with hypospadias (Fig. 2). It was corrected by Duplay's method, modified only by extending the end of the transverse incision in the skin anterolaterally along the ridges which marked the transition between penile skin medially and scrotal skin laterally. Fig. 3 demonstrates that perfect straightening has been obtained in one stage despite the extreme deformity; the ventral surface of the penis is covered by hairless skin from which it is intended later to construct a new urethra by the method of Thiersch and Cecil.

SUMMARY AND CONCLUSIONS

1. The single stage plastic operation of Duplay, devised in 1874, is, if properly performed, adequate for complete straightening of the most extreme degrees of ventral curvature occurring with hypospadias.

2. Success depends, not upon the type of incision and closure of the skin, but upon complete excision of the fibrous rudiment of the corpus spongiosum urethrae, supplemented, when necessary, by freeing and posterior displacement of the intact urethra.

3. An illustrative case (from a series of twenty) is reported.

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HYPOSPADIAS

HERBERT WILLY MEYER, M.D., NEW YORK, N. Y.
(From the Lenox Hill Hospital)

HERE are presented two cases of penoscrotal hypospadias in which the patients were operated upon nine and five years ago, respectively. The first patient was operated upon in 1930 shortly after McWhorter had published the method he employed successfully.[1] This method was based upon principles devised by Russell and published in 1900.[2] Thompson improved upon this method and in 1917 published his procedure.[3]

Operations for severe hypospadias are notorious for the difficulty in effecting a cure. Innumerable procedures have been devised and can be found in textbooks and in the literature. The late results were usually so uniformly poor that many surgeons abandoned operative therapy.

The first of our two patients was presented as a cured case before the New York Surgical Society in the fall of 1933, and the case was briefly described in the *Annals of Surgery* in 1934. In 1933 Lyle[4] published a method which had been devised by the French surgeon Ombrédanne. This operation is called the Ombrédanne pouch operation. It is a splendid method and at the time of our first operation was not known in the United States. It gives such excellent results that I believe it has been generally accepted by surgeons. Having obtained a most satisfactory result, however, in our first case by McWhorter's method, we naturally handled a second similar case by the same method, eight months previous to the appearance of Lyle's paper. This second patient has also obtained a satisfactory result and therefore it seems permissible once more to draw attention to the procedure which was employed in these two cases.

The operation is somewhat more complicated than the Ombrédanne pouch operation. The latter does not require a preliminary suprapubic cystotomy during the plastic repair. The advantage is partly effaced in my opinion by the fact that the final cosmetic and esthetic results are superior in the McWhorter procedure as compared to the Ombrédanne pouch operation. Certainly in some patients this is a point well worth considering. McWhorter advises a perineal urethrotomy in place of a suprapubic cystotomy. From experiences obtained in our two cases, I prefer the suprapubic cystotomy.

TECHNIQUE OF OPERATION

The operation is divided into two stages. The first stage consists in the release of the bent and bound-down penis and division of the fibrous bands holding it down in nature's attempt to bring the abnormal urethral opening at the base of the scrotum to the tip of the penis

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(Fig. 1). A transverse incision must be made just below the glans penis as shown in Fig. 2. The fibrous bands are completely divided so that the penis can be extended. This transverse incision is sutured longitudinally, bringing *A* to *B* at the center of the defect. This then forms a normal shaft of the penis. The result of this procedure in the second case is shown in Fig. 9. Upon the completion of this first operation, the urethral opening is approximately 2 to 2½ inches from the tip of the penis.

Fig. 1.—Penoscrotal hypospadias. Probe has been placed in urethral opening.

Following this procedure one must wait a long time until all post-operative induration has completely subsided and the thick skin of the penis is again soft, pliable, well-nourished with blood, and otherwise entirely normal. This takes many months, and the longer one waits, the better is the prospect of success in the second stage of the procedure.

The second stage constitutes the construction of a new urethra. At this time a suprapubic cystotomy is performed as a preliminary procedure. The new urethra is a tube lined with skin, fashioned from the shaft of the penis and prepuce, which will connect the cutaneous opening of the urethra with the tip of the glans where the urethral opening normally should be. Part of this newly formed tube is buried subcutaneously and the distal part is buried within the shaft of the penis to emerge normally at the tip of the glans.

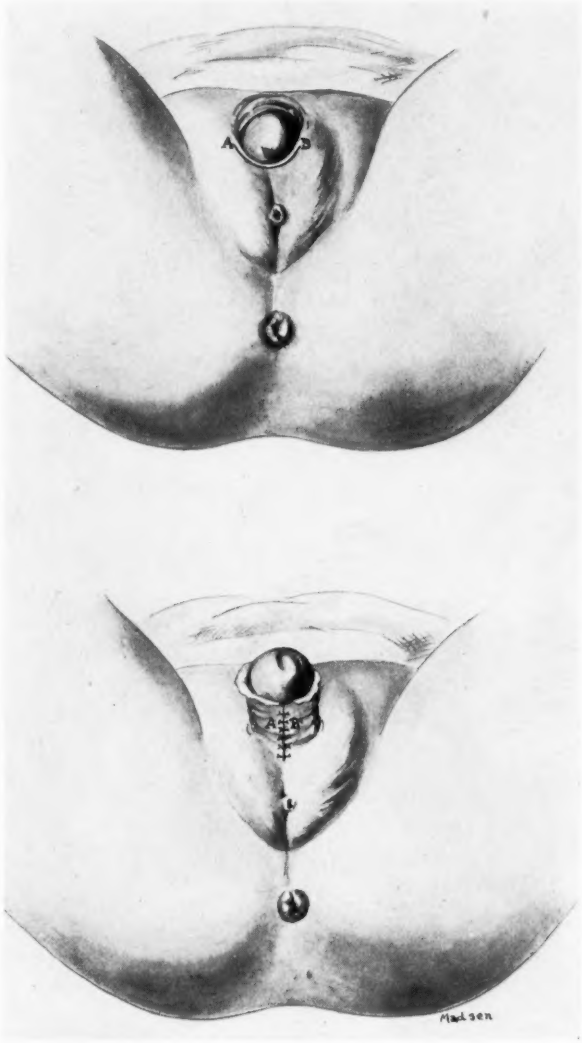

Fig. 2.—Diagrammatic drawing showing transverse incision made to free bound-down penis. *A* is brought to *B* in suturing the transverse incision vertically in order to form a shaft of the penis.

As shown in Fig. 4, a collar of skin is fashioned from the redundant prepuce, about 1 to 1.5 cm. wide, leaving it attached at the undersurface of the penis. This is lifted over the glans of the penis. The further lines of incision are parallel and run posteriorly to a point about

1 cm. behind the urethral opening. Here they are joined transversely. The incisions are deepened until they pass through the thin skin and are then dissected mesially along the shaft of the penis for a short distance only, leaving enough skin attached to the penis to furnish blood supply for the raised flaps. The remaining attached skin thereby forms the bed of the newly formed tube, while the raised portions form the lateral walls and roof of the urethra.

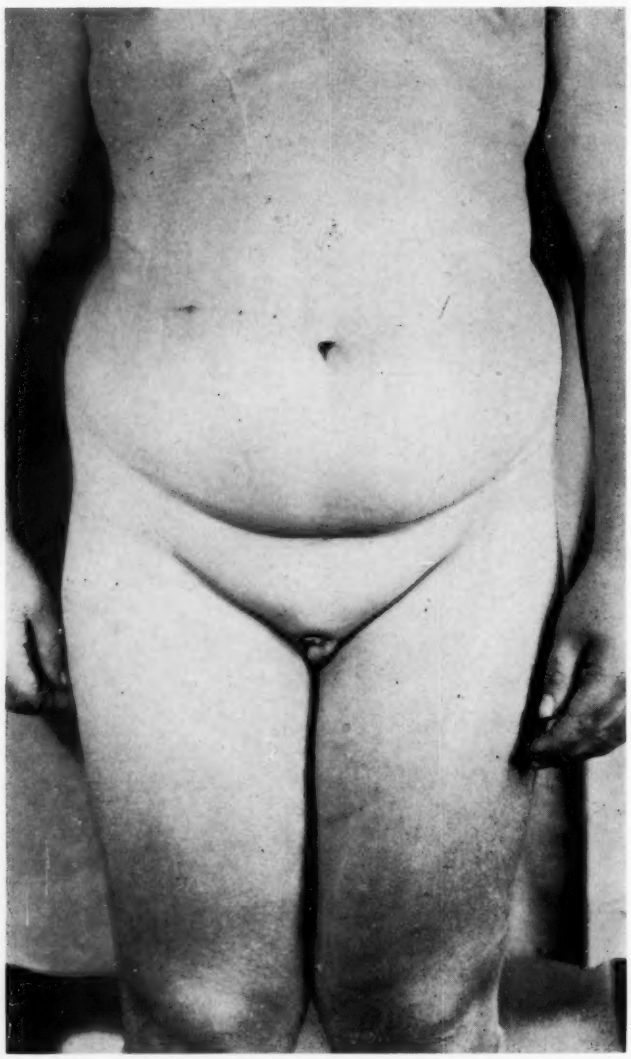

Fig. 3.—Case 1 before operation, showing undeveloped penis bound down. Note right undescended testicle.

By starting posteriorly and most meticulously suturing skin edge to skin edge with the knots inside, a tube is formed with skin on the inside and raw surface on the outside, slowly covering over and enclosing the urethral opening. The skin is, of course, very delicate (almost as thin as tissue paper) and is easily damaged and torn.

In the first case we used fine needles and No. 000 catgut. No. 0000 catgut would have been better. From experience in this case and trouble encountered in the convalescence by the extrusion of some of the catgut knots, I advise the use of the finest needles and finest silk, such as are used in blood vessel surgery. This suture material comes

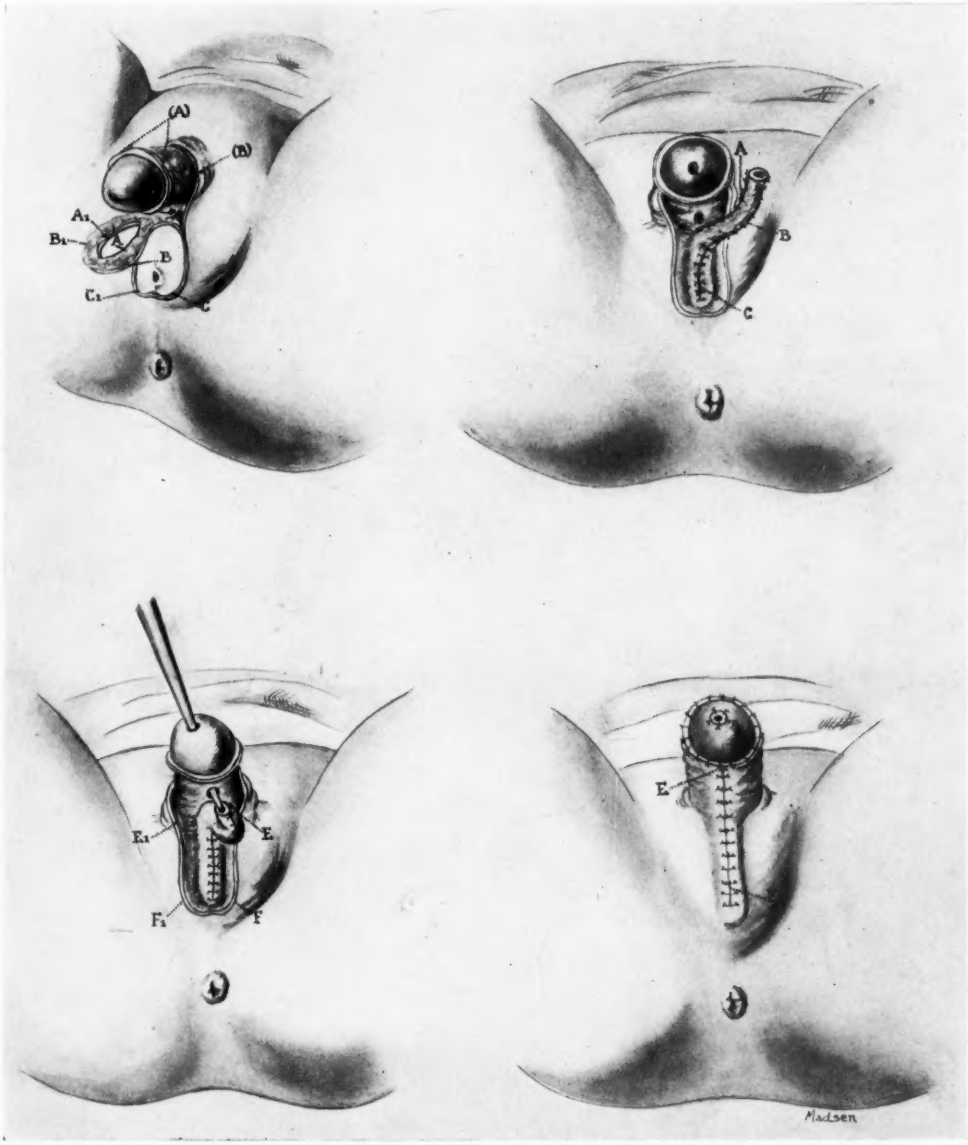

Fig. 4.—Showing the steps used to form the tubed urethral channel with skin on the inside. This tube as shown is drawn through the shaft of the penis and is then buried under the skin sutured over the tube.

all prepared and makes the smallest openings in the skin and allows better skin edge approximation.

The central opening in the collar of skin lifted off the prepuce is also sutured, thereby making an entirely closed canal and tube. At a

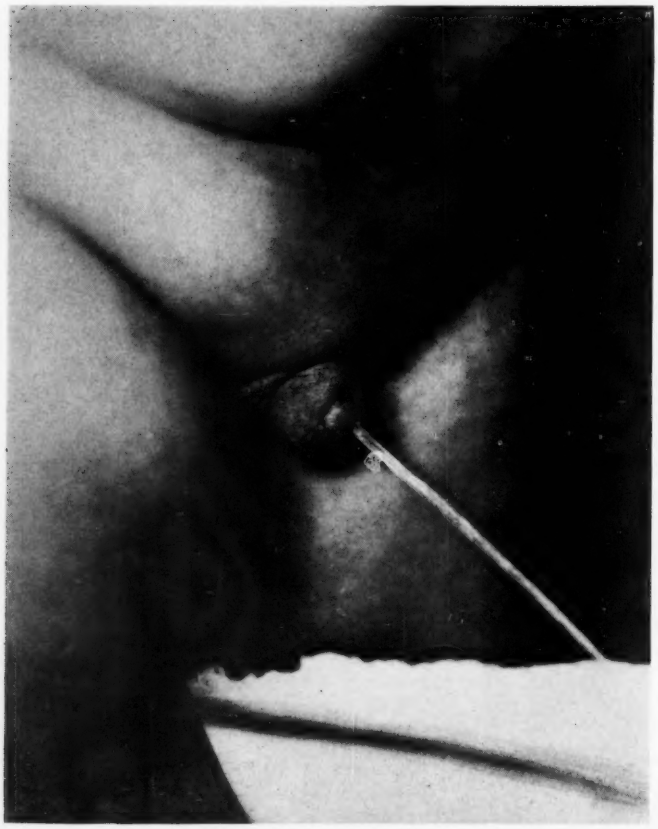

Fig. 5.—Case 1 at end of operative repair. Shows excellent urethral stream emerging normally from tip of penis.

Fig. 6.—Same patient as shown in Fig. 5 nine years after operation at age of 18 years. Note that right testicle has descended. All functions perfectly normal.

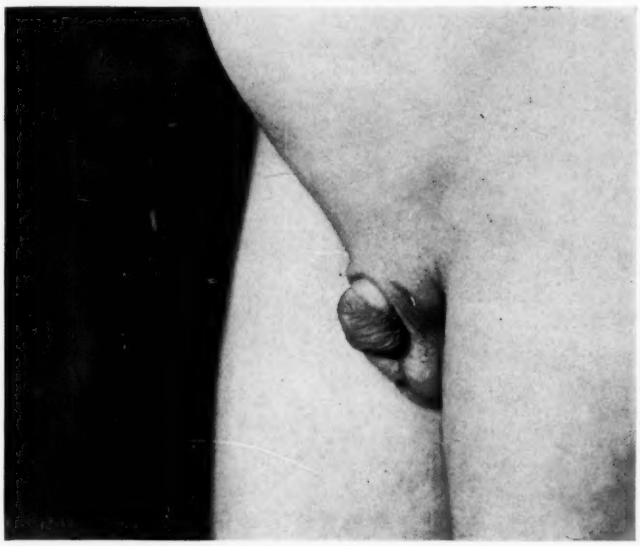

Fig. 7.—Case 2, showing the penis bent and bound down.

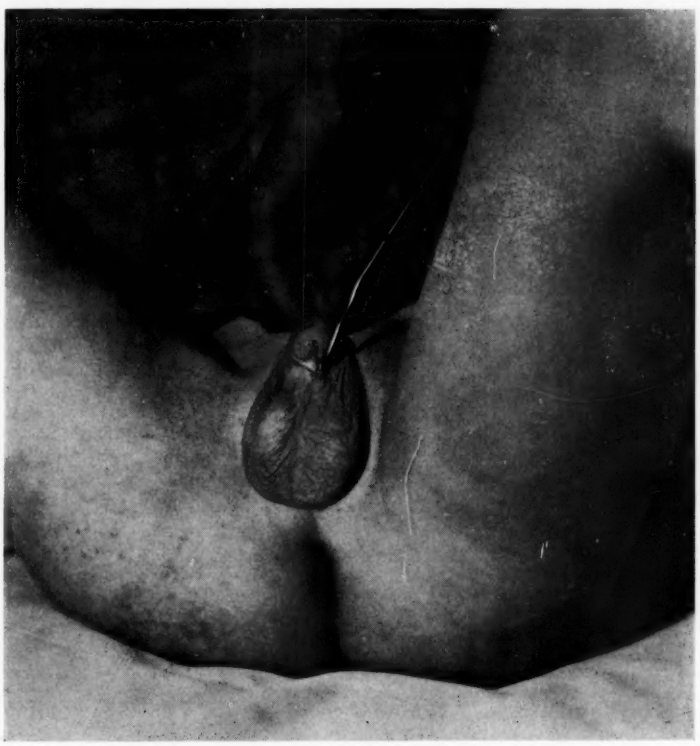

Fig. 8.—Case 2, showing probe placed in urethral opening. Note how nature tries to bring urethral opening to tip of penis.

Fig. 9.—Case 2, after first-stage operation. Shaft of penis has been formed, thereby bringing urethral opening formerly near tip of penis back at the bottom of shaft of penis at base of scrotum.

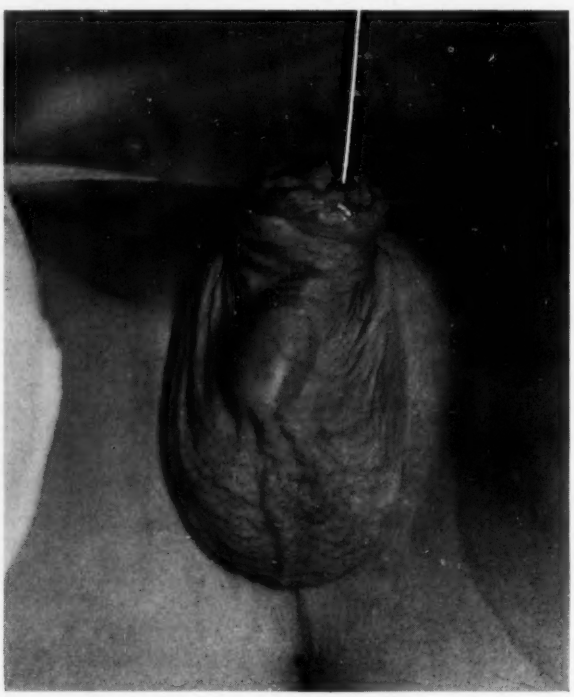

Fig. 10.—Case 2, at end of second-stage operation when urethral tube has been formed and buried under skin. Urethral sound has been passed into urethral tube all the way to the end, where urethra opens into newly formed tube.

point where this tube completely separates from the shaft of the penis, a canal is made through the shaft of the penis by plunging a narrow-bladed scalpel from the tip of the glans between the corpora cavernosa to the undersurface of the shaft of the penis, just in front of the attached portion of the newly formed tube. This canal is stretched with a small hemostat and the tube is pulled through this canal so that it emerges at the tip.

This urethral tube must now be buried and covered over with skin. This is done by undermining the lateral skin margins slightly and suturing these skin flaps across the tube with a most meticulous suture line in the center of the shaft of the penis. If there is any tension on the suture line, lateral or dorsolateral, relaxing incisions must be made through the skin of the shaft of the penis so that primary union of the

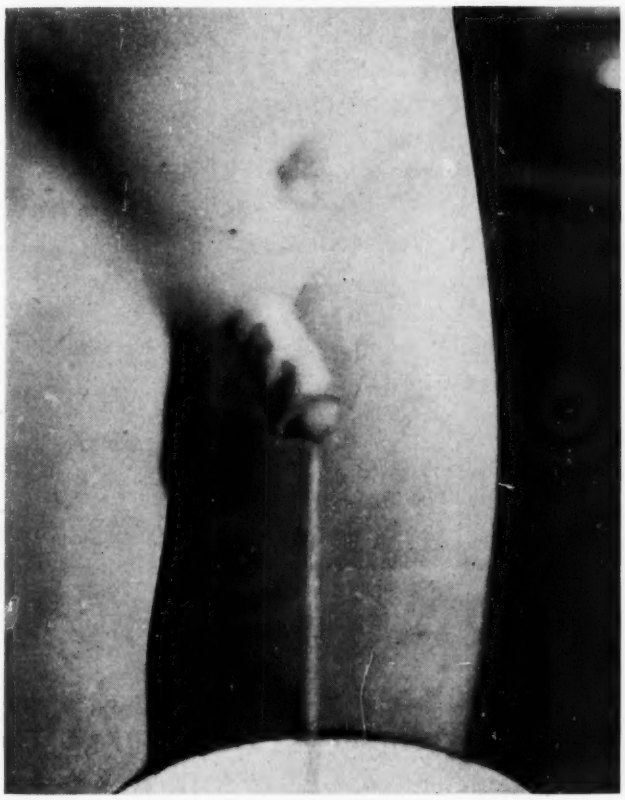

Fig. 11.—Case 2, showing excellent shaft of penis with stream of urine coming normally from tip of penis.

suture will ensue. These relaxing incisions will also help to avoid postoperative edema. The skin tube is allowed to emerge slightly beyond the surface of the glans and is cut obliquely to avoid circular stricture formation. A few sutures through the glans and tube edge will hold the tube in place and prevent it from drawing into the shaft of the penis.

The circular denuded collar of the prepuce is now sutured just the same as one does in an ordinary circumcision. Detail and exactness in suture and gentleness in handling the tissues with the finest of instruments are vital for success in healing.

The suprapubic fistula should be used for fully twelve to fourteen days before any attempt is made to allow the patient to void or until any disturbance in wound healing has been completely overcome. If there should be any leak due to improper healing, a revision is required at the site of the leak. It is important, therefore, that the suprapubic fistula be maintained until one is sure of a successful outcome. In the aftercare it is wise to pass sounds through the skin tube for the first year or so at biweekly intervals in order to make sure that no stricture develops in the urethral tube (Fig. 10).

In the first case it is now nine years since operation. The young man has grown to manhood and every accompanying function has developed normally. The right undescended testicle (Fig. 3) suddenly descended into the scrotum during an attack of severe pain following some injections of antuitrin S. The urinary stream is perfectly normal (Fig. 5). Fig. 6 shows the present absolutely normal appearance of the external genitals.

In the second case it is five years since operation and this boy also is normal in appearance and function, but he is still in the adolescent period (Figs. 7-11).

Granting that the Ombrédanne pouch operation is possibly the simpler procedure with a greater probability of early success and shorter convalescence than the McWhorter type of operation, I still feel that the successful outcome of the cases described here warrants the addition of this procedure to the surgical armamentarium. Failure to cure a patient with marked hypospadias will only lead to unhappiness and inferiority complexes for the unfortunate individual.

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AN IMPROVED APPARATUS FOR TIDAL DRAINAGE OF THE URINARY BLADDER AND EMPYEMA CAVITIES

CARROLL J. BELLIS, M.D., MINNEAPOLIS, MINN.
(From the Department of Surgery, University of Minnesota Medical School)

IRRIGATION OF THE URINARY BLADDER

NEUROGENIC Vesical Dysfunction.—Urinary sepsis, originating in the bladder and urethra and extending into the upper reaches of the urinary tract and gradually destroying the kidneys, is the most common and fatal sequel to spinal cord injury. The extending infection is directly attributable to either (a) repeated instrumentation of the urethra coincident with repeated catheterization or (b) interruption of the neurophysiologic cycle of intravesical pressure and urinary expulsion.

The accepted teachings of Sherrington[1] assign a postural tone to the normal detrusor muscle, permitting an isotonic filling of the bladder to a pressure of from 6 to 10 cm. water. When the intravesical tension is from 10 to 14 cm. water, desire to void is felt. The tension has been shown cystometrically to rise rapidly from that point so that, when the bladder contains from 200 to 400 c.c., a pressure of from 20 to 30 cm. water exists.

Neurogenic vesical dysfunction results from lesions of the brain, peripheral nerves, spinal cord, or of the intramural ganglia through injury or destruction of the centers for bladder function or of their afferent or efferent pathways. This occurs in central nervous system lesions such as tabes dorsalis, cord tumors, cord trauma, multiple sclerosis, subacute combined degeneration, syringomyelia, spina bifida, and infectious and traumatic myelitis. It is also seen in bilateral frontal lobe and caudate nucleus lesions. In neurogenic vesical dysfunction, desire to void is not felt until the intravesical pressure is higher than that normally adequate (Creevy[2, 3]). The resulting constant elevated pressure leads to capillary stasis in the bladder wall and atrophy of the detrusor muscle with retention through lack of expulsive force. At abnormally high intravesical pressures, bladder contractions expel urine without the attending desire to void, this state being known as paradoxical or overflow incontinence. The retained urine causes an additional pressure anesthesia of the bladder wall augmenting the degree of retention.

Creevy has recommended the use of conservative measures for the treatment of neurogenic vesical dysfunction. These are (a) voiding "by

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the clock'' at regular and frequent intervals, (b) cholinergic drugs (eserine, pilocarpine, and acetylbetamethylcholine chloride), and (c) a combination of (a) and (b) with intermittent catheterization. Where complete bladder rest is desired, it is accomplished by cystostomy. In addition, he[4] has pointed out that mechanical obstruction to urination so slight as to cause no symptoms in the normal bladder may lead to retention or overflow if only mild neurogenic dysfunction exists.

Cystitis.—Where the bladder has become secondarily infected due to an indwelling catheter or as a sequel to radium therapy for carcinoma of the cervix with a succeeding Wertheim operation, frequent irrigation of the bladder at low pressures is imperative. Often, however, cystitis is present in association with carcinoma of the cervix prior to therapy. Bladder infections produced through these or hematogenous agencies may be effectively treated by specific peroral chemotherapy where the etiological bacteria are identified. In addition, the local application of these antiseptics is beneficial when they are applied as irrigants. Regular frequent irrigations assist in removal of secretions and reapplication of fresh antiseptic solution.

THORACIC EMPYEMA

Carlson[5] and Carlson and Bowers[6] have emphasized that five principles govern the treatment of thoracic empyema:

1. Adequate drainage.
2. Avoidance of early open pneumothorax.
3. Rapid sterilization of the diseased area.
4. Early obliteration of the cavity by expansion of the lung.
5. Good medical care and nutrition of the patient.

In the meta- or postpneumonic types of thoracic empyema, aspiration is continued until the pus is too thick. Then dependent drainage is established through an intercostal catheter. Frequent irrigation of the empyema cavity facilitates the free drainage of pus, washes and sterilizes the cavity walls, and digests the exudate on the visceral pleura which restrains the lung in its natural tendency to re-expansion (Wangensteen[7]).

The use of a water-sealed suction at a negative pressure of from 10 to 30 cm. water to re-expand the lung has been stressed by Wangensteen. Where the pleura is thick, adequate drainage by suction alone cannot be obtained. Dilution of the exudate by an irrigating fluid renders the negative pressure more effective. Plugging of the intercostal catheter requires washing out of the catheter in situ or replacement. Heretofore, it has been the practice to manually irrigate and aspirate the intercostal catheter at frequent intervals.

APPARATUS

Hart[8, 9, 10] and Davis[11] were among the first to suggest useful decompression and irrigating devices for thoracic empyema and the urinary

bladder. Recently, Myers[12] has described an automatic bladder irrigator which delivers a definite quantity of irrigant to the bladder, regardless of the intravesical pressure. Vary[13] has described a somewhat complicated tidal irrigator which has many functional advantages. Munro and Hahn,[14, 15] however, designed an excellent bladder tidal irrigator

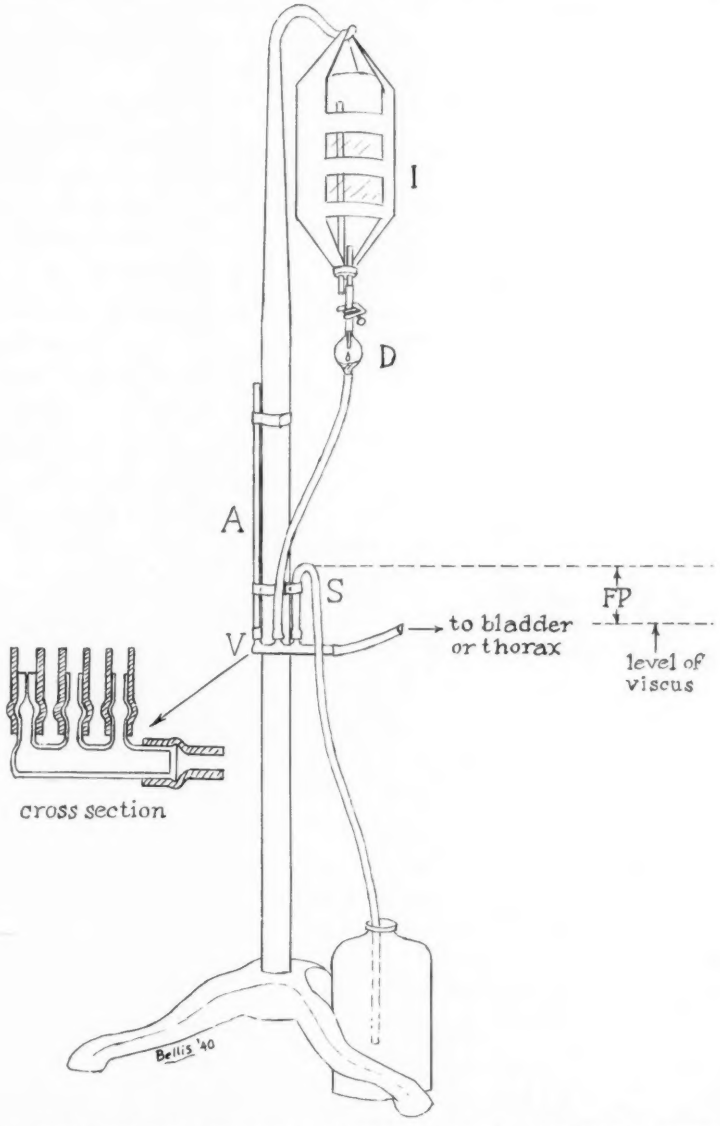

Fig. 1.—Tidal drainage apparatus. *I*, irrigant; *D*, drip regulated by screw clamp; *FP*, filling pressure at which siphon, *S*, will trip; *A*, air vent and manometer; *V*, valve made of fused outlet of glass Dakin's tube.

pre-eminently for use in spinal cord injuries. Through prompt use of this apparatus, they were able to reduce urinary sepsis in cord injuries from 73 to 15 per cent. The accurate adjustment of the component parts of their apparatus, however, and the difficulties often accompanying its proper function have often been troublesome.

In its place, the apparatus shown in Fig. 1, in use in the University of Minnesota Hospitals, is suggested for tidal irrigation of the bladder or empyema cavities. The irrigant (*I*) is placed in a suitable reservoir at least 75 cm. above the level of the cavity to be irrigated. It is delivered at the rate, usually, of from 40 to 60 drops per minute, the delivery being increased or decreased according to the frequency of desired drainage periods. The flow is regulated through the drip (*D*) by a screw clamp; the drip must not contain an air vent. Entering one vertical arm of a three-outlet glass Dakin's tube, the solution passes through the horizontal arm to the indwelling catheter. Through the other vertical arms, the irrigant is diverted, respectively, through a siphon (*S*) of flexible rubber intravenous tubing and a vertical glass tube (*A*) about 0.6 or 0.7 mm. in diameter which serves as an air vent and manometer. At the bottom of the air vent, a valve (*V*) is interposed. This consists simply of one of the glass Dakin's tube outlet tips which has been fused to a capillary of a diameter just sufficient to admit the point of a common pin. The height of the siphon is adjusted to the desired pressure (*FP*) at which the bladder or empyema cavity is to be filled to initiate siphonage by fastening its upper curve at the desired height above the bladder or intercostal catheter. The level of the pubis and the point of entrance of the intercostal catheter into the chest are respective convenient landmarks for zero points. The distal portion of the siphon empties into an open drain bottle.

The process in the ordinary course of events consists in a simultaneous flow of irrigant into the bladder or chest and up the proximal arm of the siphon to the level of the filling pressure at which the arch is set, and through the valve into the vertical air vent. When fluid overflows the arch, siphonage is instituted, rapidly draining the bladder or pleural cavity. However, fluid is siphoned from the air vent very slowly, due to the interposed valve. The viscus is thus emptied before the siphon is broken by air coming through the vent. By adjusting the rapidity of flow through the drip or the filling pressure, each cycle may be made to vary from a few minutes to one or two hours. The apparatus is functioning properly when respiratory excursions are visible in the air vent or in the proximal arm of the siphon. Where suction is to be instituted in thoracic empyema, the irrigator is discontinued, and the suction outlet is connected to the intercostal catheter.

A "reservoir," wasting its volume of irrigant at every cycle, is thus eliminated. In addition to the few connections, it is not necessary to give extraordinary attention to the size of the tubing. The opening in the valve must be so small that the viscus will empty before the vertical glass air vent is cleared during siphonage. The rubber connections are of ordinary intravenous tubing. If no glass Dakin's tubes are available, T-tubes of usual hospital or laboratory sizes may be used, and the glass portion of an eye dropper may be fused as a valve.

For the bladder the irrigant is ordinarily saturated boric acid solution, but may be replaced by silver nitrate, potassium permanganate, or other antiseptics for tidal irrigation of the infected bladder. In the latter case, only low intravesical pressures should be used, the siphon arch being elevated to not more than from 4 to 5 cm. above the pubis. For pleural irrigations, physiologic saline solution or Dakin's solution is commonly used at a filling pressure of less than 10 cm. water.

DISCUSSION

The common term "cord bladder" refers to a bladder which functions abnormally due to disease or injury of the spinal cord. This state is properly called neurogenic vesical dysfunction, and, in its management, tidal drainage diminishes the urinary sepsis attending the use of the indwelling catheter. According to Munro, these bladders pass through three definite stages if the lesion is above the sacral segments. Hematomyelia or edema at any level or incomplete cauda equina lesions introduce a fourth stage.

1. The *atonic* bladder is due to an areflexia occurring immediately after injury to the cord during the period of spinal shock. The capacity of the bladder allows extreme distention, the external sphincter being under neither voluntary nor reflex control. Tidal irrigation at an intravesical pressure of from 2 to 5 cm. water is recommended here.

2. The *autonomous* bladder invariably follows the first stage, and, in addition, is the end stage in complete destruction of the sacral cord or cauda. Despite the persisting external areflexia, the intramural reflex control of the detrusor muscle and internal sphincter allows intermittent emptying with a storage capacity less than that of the atonic bladder. Central inhibition is, of course, absent here, and tidal irrigation at an intravesical pressure of from 10 to 15 cm. water is recommended.

3. The *hypertonic* bladder is manifest in all cord injuries except those of the sacral segments and cauda. The segmental spinal reflex activity has been re-established in an uncontrolled manner, the bladder emptying at irregular intervals. The storage capacity is less than that of the autonomous bladder.

4. The *uninhibited reflex* bladder operates independent of central inhibition of which it has been deprived. It empties incompletely through a local stretch reflex, the retention favoring a tendency toward total reversion to the *atonic* bladder.

The differential diagnosis of these types is made by cystometry, the cystometrograms being easily made with the apparatus described herewith and compared with the normal curve. By varying the intravesical pressure of the tidal irrigator as the bladder passes through the stages outlined, urinary stasis is obviated and the infection consequent to the indwelling catheter circumvented.

In thoracic empyema, adhesions forming between the visceral and parietal pleura contribute resistance against collapse and mediastinal shift produced by the positive pressure of the atmosphere. When this stage has been reached, signalized by the changing of thin pus to highly viscid exudate, intercostal catheterization or open drainage may be safely instituted. We have found such pus to contain from 50 to 80 per cent protein.

Tidal irrigation assists in diluting the thick pus and in its removal. It contributes to sterilization of the cavity by frequently washing away secretion and introducing antiseptics. Only in this manner must it be considered as aiding in re-expansion of the lung. In a somewhat similar manner, blowing exercises aid in re-expansion of the lung. The increased intrapulmonic pressure attending blowing against resistance has no effect whatever upon the intrapleural pressure of the thoracotomized side. Its only asset lies in the fact that during the act it maintains the lung distended while the contraction of the thoracic expiratory muscles decreases the volume of the pleural space and forces pus through the thoracotomy. Thus, blowing exercises have their only virtue in very slightly stretching adhesions during the preliminary deep inspirations, facilitating mobilization of the lung. Drainage, irrigation, or blowing exercises are virtually ineffective in re-expanding the lung, a task effectively discharged by continual suction applied to the indwelling thoracic catheter.

SUMMARY

The effects of neurogenic vesical dysfunction upon the emptying mechanism of the bladder are described insofar as the physiology of micturition is known. The neurophysiologic alterations of the detrusor muscle and sphincter control attending spinal cord injuries are briefly reviewed. The effects of irrigating thoracic empyema cavities are enumerated, and the advantages are discussed. A simplified apparatus for tidal irrigation in neurogenic vesical dysfunction, for irrigation of the infected viscus with undisturbed central control, or of thoracic empyema cavities is described.

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PERINEAL PROSTATECTOMY

A Modification of Closure

Samuel A. Vest, M.D.,* University, Va.

(From the James Buchanan Brady Urological Institute, Johns Hopkins Hospital, Baltimore, Md.)

In a recent publication[1] I have reported a series of 365 consecutive prostatic operations with a mortality of 1.6 per cent. It was felt that this mortality was particularly significant because the patients were on a public ward and a good percentage were unusually poor candidates for any type of operative procedure. The average age was well over 60 years. It was further significant that the operations were performed entirely by the resident house staff, who were receiving their first operative training and experience. It was pointed out that perineal prostatectomy, suprapubic prostatectomy, and transurethral removal of the obstructing prostate each has its place and that, if the indicated operation in each individual case is carried out instead of a stereotyped procedure, the maximum satisfactory results would be obtained. In general, the size of the obstructing prostate was the determining factor in the selection of either open operation, perineal or suprapubic, or transurethral, using the original Young punch or Thompson's modification, or the McCarthy resectoscope. In the treatment of patients presenting the average size or above prostatic hypertrophy, prostatectomy was carried out in 233 of the 365 cases and in practically all instances this was perineal prostatectomy.

The advantages of removing medium- and large-sized adenomas by the perineal route, such as visualization of the operative field with satisfactory control of hemorrhage, dependent drainage, putting the bladder at rest to minimize upper tract infection, etc., have often been stressed by Young[2] and others. The results published by us recently from the ward service have again served to emphasize the advantage of the perineal removal of enlarged prostates according to the technique of Young as an extremely valuable procedure in a large percentage of cases.

It is the purpose of this publication to present a modification of closure in perineal prostatectomy which I found advantageous in certain instances during my tenure as resident in the Brady Urological Institute.

One of the most formidable complications of surgical removal of the prostate, to be avoided whenever possible, is postoperative hemorrhage. Postoperative hemorrhage following perineal prostatectomy for obvious reasons is the least likely complication as compared with either supra-

*Lalor Fellow in Urology, Johns Hopkins University.

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pubic or transurethral procedures. Continued hemorrhage following transurethral resection with blockage of the catheter by clots results in distention of the bladder, and technical procedures to aspirate and free the bladder of blood may result in infection, pyelonephritis, and other serious complications. Continued hemorrhage following either perineal or suprapubic prostatectomy may also sometimes necessitate aspiration and irrigation and even reopening the incision and insertion of packs into the vesical orifice. Such a complication may be followed by shock, or local infection of the wound or bladder with delayed healing and actually acute or subacute pyelonephritis and septicemia. It is possible that marked hypertension with rigidity of the walls of the arteries supplying the prostate might tend to prolong bleeding following removal of the adenomatous prostate. There seems indeed to be considerable variation in the vascular supply of individual prostates. In some cases complete removal of a large adenoma by the perineal route is practically bloodless, while an identical case will show a tendency to considerable vascular oozing, necessitating suturing the vesical neck, packing of the wound, or insertion of various types of traction bags. The majority of the bleeding following perineal prostatectomy seems to come from the margin of the vesical orifice which is well illustrated in Fig. 1. Bleeding from the prostatic capsule or from the prostatic tissues is usually slight and of no consequence.

One of the main advantages of perineal prostatectomy is that the operation is carried out under vision and it is not difficult to determine the source of hemorrhage. I have yet to encounter any hemorrhage which has not been readily controlled at operation except that from the margins of the retracted vesical orifice. By use of proper methods, one of which is described here, hemorrhage following perineal prostatectomy can be kept to a minimum or entirely controlled. In the recent report referred to above, transfusion because of postoperative hemorrhage was necessary in only 4.7 per cent of the cases following prostatectomy.

The prevalent method of controlling hemorrhage following prostatectomy on the ward service six years ago was either by packing the vesical orifice with gauze or by means of a Davis traction bag. These methods have gradually been abandoned in favor of simple sutures into the vesical orifice so as to approximate the vesical orifice to the prostatic capsule. Statistics on the ward service in about 500 cases have shown that in the hands of the residents there have been distinctly better results with less complications and shorter healing time of the perineal wound if the vesical orifice was sutured. With this apparent distinct advantage following the use of sutures, the only complication I have been able to discern has been occasional postoperative hemorrhage which has sometimes necessitated packing or insertion of a rubber bag into the wound. This hemorrhage apparently occurs from the margin of the vesical orifice and is due to failure of the type of sutures used. The

sutures for the most part have been simple sutures approximating the various quadrants of the vesical orifice to the prostatic capsule in such a way as to pull the vesical orifice against the capsule, thus simulating normal relationships. The failure of the sutures in rare instances has

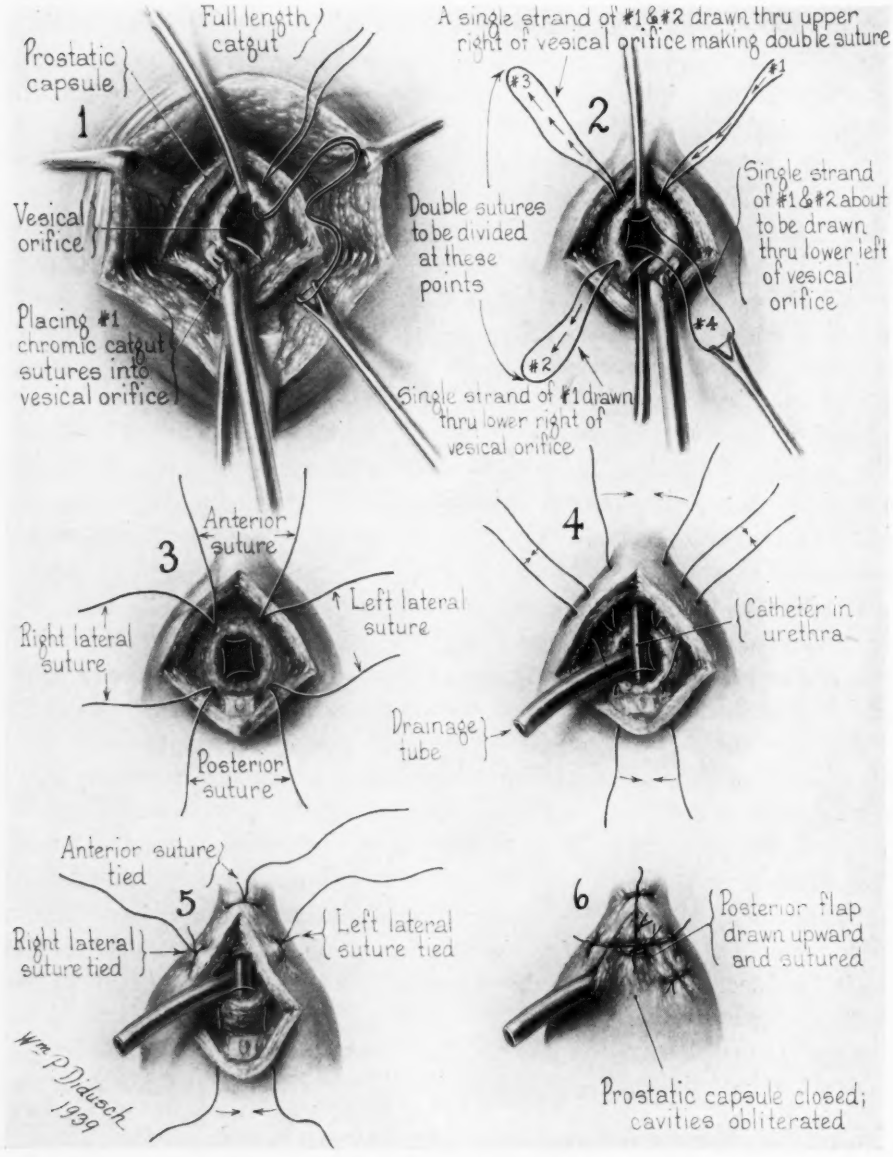

Fig. 1.—Technique of reconstructing the vesical orifice to eliminate hemorrhage following removal of benign prostatic hypertrophy.

been due to the fact that they did not circumscribe the vesical orifice in its entire 360 degrees so that, when hemorrhage occurred, it was probably due to a small spurting vessel between two sutures. With this in mind, I have modified the closure so that the full 360 degrees of vesical

orifice is included by the sutures. At the same time these modified sutures have reconstructed the vesical orifice in a manner similar to that present in the normal. A description of the technique is as follows:

Routine perineal prostatectomy, according to the technique described by Young, has been carried out in the clinic and needs no reiteration here. It should be emphasized, however, that, in order to facilitate the proper use of the sutures, the surface of the prostatic capsule should have adequate exposure and the inverted V-incision should be slightly wider and larger than usual. This makes the placing of the sutures, to be described, less difficult. Fig. 1 shows the various steps I have found advisable in cases in which there seems to be an unusual amount of vascularity at the vesical orifice in order that postoperative hemorrhage may be kept to a minimum or entirely controlled. In Fig. 1, *1* shows the prostatic cavity immediately following thorough removal of a large adenoma. The vesical orifice is pulled well into view by two long tenaculums which have grasped it anteriorly and posteriorly. The adenoma has been cleanly cut from the vesical orifice and posterior urethra by means of scissors.

By means of a boomerang needle, the long double suture of No. 1 plain catgut has been pulled through the vesical orifice in the region of 2:00 o'clock. This suture is shown with Young's boomerang needle just before grasping it and pulling the same suture through the vesical orifice in the region of 8:00 o'clock. These sutures are taken rather deeply into the vesical orifice so that they will include any retracted open vessels that might be on the bladder side. The placing of this suture may be sometimes awkward because a complete length of catgut, even though doubled, is not quite long enough. This can be remedied by tying two lengths together before beginning the procedure. In Fig. 1 (*2*) the double suture has now been pulled through the vesical orifice both in the region of 10:00 and of 8:00 o'clock, following which a single strand of the double suture which transverses the vesical orifice has been grasped and pulled through the vesical margin in the region of 10:00 o'clock and the boomerang needle is shown in the act of pulling the remaining strand across the vesical orifice through the margin in the region of 4:00 o'clock, following which the sutures are divided at the points labelled *2*, *3*, and *4*. The situation after these sutures have been cut is shown in Fig. 1 (*3*). There are shown four individual sutures at the vesical orifice which are indicated as anterior, posterior, right and left lateral. As will be seen in *3*, each strand of the left lateral suture, for example, goes through the same needle hole in the vesical orifice as the adjacent anterior and posterior suture, so that no vesical orifice margin is left between in which there might exist a spurting artery. The four sutures, therefore, include the entire 360 degrees of vesical orifice. In Fig. 1 *4* these four sutures have been pulled through the corresponding areas in the prostatic capsule, following which a

urethral catheter and a small perineal drainage tube are shown in place. It will be noted that the anterior suture has been placed far anteriorly in the prostatic capsule so that when it is tied the anterior lip of the vesical orifice is pulled down on the anterior surface of the catheter and the anterior margin of the vesical orifice will then very closely approximate that of the divided urethra. The portion of the prostatic cavity where the anterior prostatic commissure and urethra once existed is thus obliterated. The lateral sutures are next tied as shown in Fig. 1 (*5*). The last procedure is to tie the posterior suture which ligates the ejaculatory ducts. After tying the sutures, a single strand of each is divided as shown in Fig. 1 (*5*). In *6* it can be seen that the single strand of posterior suture has been tied to the anterior and the two laterals have been tied across the midline. The tying of the posterior to the anterior has carried forward the tip of the inverted flap and reapproximated it into its original position with the tube coming out one of the limbs. Sometimes a small single suture is placed on the opposite side of the V as shown in Fig. 1 (*6*) to close completely the prostatic capsule except at the point where the tube emerges. With this type of closure the vesical orifice is thoroughly circumscribed in its 360 degrees and is pulled down into the cavity of the prostatic capsule, very near to the stump of the membranous urethra. This tends to produce early obliteration of the prostatic capsule and leaves only a very short bridge for the regenerating mucosa of the vesical orifice to transverse before it meets the mucosa of the membranous urethra. The perineal tube is withdrawn at the end of twenty-four or forty-eight hours according to whether or not perineal drainage is necessary because of infection, etc. The remainder of the perineal wound is closed according to the usual technique, which consists of pulling the levator ani muscles together with plain catgut and suturing the perineal skin with interrupted waxed silk or catgut sutures.

Numerous methods for handling the vesical orifice, prostatic capsule, and perineal wound by means of sutures, all of which have undoubtedly given good results, have been reported by Young, Wildbolz, Hinman, and others. In my hands the present technique has been very satisfactory and it is believed to eliminate greatly the possibility of hemorrhage and at the same time to effect a normal anatomical reconstruction of the prostatovesical relationships. I have had no difficulty in inserting sutures according to the method described and the entire operation, in most cases, has been carried out in about thirty minutes. There has been no postoperative hemorrhage in any instances and this type of suture has in no way interfered with proper and rapid healing of the perineal wound. The first thirteen cases in which I used this method were included in the recent report on prostatic mortality. The average healing time for the perineal fistulas was 13.5 days, following which each patient voided freely with complete control. In one case I cysto-

scoped a patient on the eighteenth day with both the Young and McCarthy cystoscopes and found the mucosa and vesical orifice to be normal in every way except for some hyperemia. There were no denuded areas between the membranous urethra and the bladder and entire healing of the mucosa of the prostatic urethra had taken place by the eighteenth day. The procedure has been carried out a number of times in private cases with entirely satisfactory results and it is advocated as a valuable method following perineal removal of the benign prostate in cases where there seems to be an unusual amount of vascularity and tendency to hemorrhage.

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THE PRINCIPLES AND THE PRACTICE OF THE RADICAL OPERATIONS FOR HERNIA

PROF. DR. EUGENE PÓLYA, BUDAPEST, HUNGARY

I. GENERAL PRINCIPLES

THE main purpose of every radical operation for hernia is to restore the integrity of the abdominal wall and its resistance in places where it is likely to yield to intra-abdominal pressure. There are generally defects of the musculature or the aponeurotic structures, or both, where the resistance of the abdominal wall to intra-abdominal pressure is impaired. Indirect inguinal hernias not infrequently deviate from this rule. The muscles are well developed and the aponeurosis faultless so that even large hernias of this kind can be cured by high ligature and removal of the sac alone (Kocher's method). Frequently, however, one finds weak muscles and an unsatisfactory aponeurosis even in congenital hernias, and then cure by simple ligature of the sac is impossible. In these cases of indirect inguinal hernia the reconstruction and strengthening of the abdominal wall is as necessary as it always is in direct inguinal and in all other forms of hernias.

The closure of the defect of the abdominal wall is undoubtedly the most important part in the radical cure of the hernia, but the extirpation of the hernial sac and its high ligation must generally precede it. However, the latter procedure may be unnecessary in cases in which there is only a diffuse protrusion of the peritoneum. In such cases even the opening of the peritoneum can be avoided, but the protruding peritoneum must be carefully freed of all its adhesions, and the margins of the hernial defect of the abdominal wall clearly exposed before its closure is begun.

The closure of the abdominal defect ensures radical cure only when it is complete and when the permanency of the broad contact of the structures united by the sutures is assured.

In order to do this, the following rules must be constantly regarded:

1. Avoid all tension when suturing.
2. Imbricate the layers which close the defect.
3. Take care that the weak places in different layers do not overlap each other, but are covered quite reliably by other layers.

II. THE IMPORTANCE AND THE MEANS OF RELIEVING TENSION

The avoidance of all tension and stretching of tissues when uniting them with sutures is the first rule for the radical cure of hernias; without this one cannot hope for permanent results. Keeping this rule is the principal condition for the strong and permanent union of the structures which are united by the sutures.

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There is a general false belief that the different structures united during the operation are held together by the sutures. Whereas this may be true for a short time after the operation, the permanency of the condition created by the operation is ensured only by the scar tissue which firmly unites the structures which were sutured to each other. This scar formation surely takes time, and the sutures must keep the united structures in contact while this occurs. If the sutures are torn or become too loose before strong adhesions between the united structures are formed, disunion of the suture line and recurrence may result. However, it should be realized that, even if the sutures are not resorbed and not torn or the knots untied, they do not constantly keep the tissues as tightly together as they did immediately after the operation, because they cut through. The greater the pressure they exert on the tissues, the earlier and the more likely will this occur, because the greater will be the extension of the aseptic necrosis which every stitch produces in the living tissue. Therefore, the greater the stress when tying a suture, the greater the probability of its early loosening or complete cutting through, even under perfectly aseptic conditions. Thus, care must be exercised to avoid too great tension upon tissues, too much when uniting them, and too great force when tying the sutures.

The stretching of the tissues and the pulling of the sutures can be avoided only if there is no tension, when the structures which should be united can be brought easily into contact with each other. This is desirable for the broad contact of the tissues which must be coaptated. This is a very important safeguard for the security and permanency of the cure. It not only prevents the loosening of the sutures, but it also eliminates the bad consequences of such an occurrence and ensures a broad and firm adhesion of the structures which were brought into contact by the operation. This is of particular importance when fascias or aponeuroses are to be sutured. They are not only very thin, but also have a very scanty blood supply and therefore the approximation of their edges with each other or with any other structure results in a thin and weak scar. Only the coaptation of broad surfaces may produce a reliable union. This can be done by mattress sutures or simple sutures, which fasten one margin of the wound to the surface of the aponeurosis at some distance from the other margin of the wound (Fig. 1).

The degree of tension in different cases and types of hernias is dependent on various factors:

1. The general condition; i.e., the tenseness or flabbiness of the abdominal wall. The flaccid abdomen of a woman who has had several children often allows easy closure of such defects which would present great difficulties if the patient were a man or a young girl.

2. The localization of the defect. Defects of large size present generally much greater difficulties if they are localized in the epigastrium

than anywhere else. This is caused by the insertion of the broad abdominal muscles on the ribs. These are very near the epigastric defects and hinder the stretching of the aponeurotic structures of this region, which consist only of the tendons of the external and internal oblique and the transverse muscles.

3. Individual conditions depending chiefly upon the size and the form of the defect and the character of its surroundings.

a. Other things being equal, the larger the defect, the greater the tension, but other factors are generally of more importance and if these are favorable very large defects often can be closed very easily and without any stretching.

b. The form of the defect is of importance in deciding upon the technique of closure because the tension of the tissues varies in different directions and by choosing the right direction for coaptating the tissues one may lessen the difficulties considerably.

c. The condition of the tissues surrounding the defect may influence their usefulness as plastic material as well as the possibility of their mobilization which may be required to overcome tension.

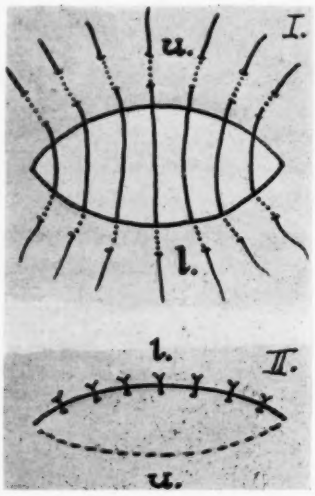

Fig. 1.—Duplication of the aponeurosis. *u*, Upper margin of the wound of the aponeurosis; *l*, lower margin; after tying the sutures the upper margin is covered by the lower one. *I*, Sutures put in; *II*, sutures tied.

III. MEANS OF OVERCOMING TENSION

This can be done principally in two ways: (1) by satisfactory mobilization of the tissues and (2) by choosing the correct method for their coaptation.

Because the latter may save unnecessary dissection and waste of tissue, it may be considered first. Different tissues bear different degrees of stress. There are also differences in the tissues which may be used for closing hernial defects, depending upon different types of hernias. There are also individual differences in the same kinds of hernias. These may be very conspicuous, especially in recurrent hernias and in

incisional hernias which all develop in postoperative scars and may therefore require individual measures.

But there are also some general rules related to the forms of the defects and these should be discussed now because they are founded on purely geometrical and physical considerations.

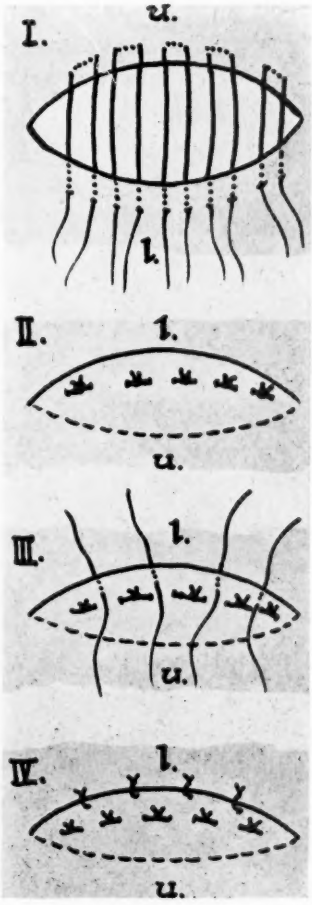

Fig. 2.

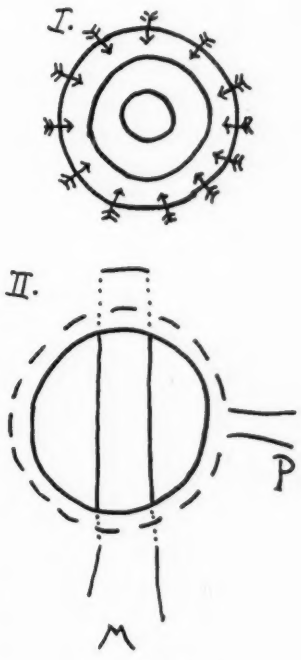

Fig. 3.

Fig. 2.—Duplication of the aponeurosis when there is a longitudinal defect with acu e angles. *u*, Upper margin of the wound; *l*, lower. The duplication of the linea alba is performed in the same way; then the margins of the wound lie naturally to the right and the left. *I*, Putting in the mattress sutures; *II*, after tying the mattress sutures the upper margin of the wound is covered by the lower (so may, for instance, the left by the right one); *III*, putting in simple sutures in the free margin; *IV*, tying the second row of sutures.

Fig. 3.—*I*, Widening of a little hole of the abdominal wall; the arrows show the direction of the least resistance of the tissue surrounding the widened hole; *II:P*, purse-string suture; *M*, mattress suture.

In the case in which there is a more or less linear defect, i.e., a simple slit or a relatively narrow gap whose length is much greater than its breadth and whose margins meet at more or less acute angles, the defect can be closed in the easiest way by drawing its margins together perpendicularly to the longitudinal axis of the defect. In this manner the margins meet in a more or less straight line. It is still better if

the tissues in the neighborhood of the defect are overlapped in the same direction (Fig. 2).

If, however, the defect is round, or somewhat circular, the attempt to unite its margins in the aforementioned linear manner would be the most difficult way of doing it. The opposite points would be drawn together in the direction of the chord, those in the middle of the defect in the direction of the diameter (Fig. 3-*II, M*), which is the longest chord and the greatest possible distance between any two points of the circle; but also the parts which lie at the sides of the circular defect can be united only with the greatest difficulty if a linear union is attempted. The correct method of closing these defects is based upon a consideration of their development. It is generally quite a small hole through which a small nerve or blood vessel passes, which gradually widens to a large round defect. This type of defect is encountered chiefly in umbilical or paraumbilical and in epigastric hernias,

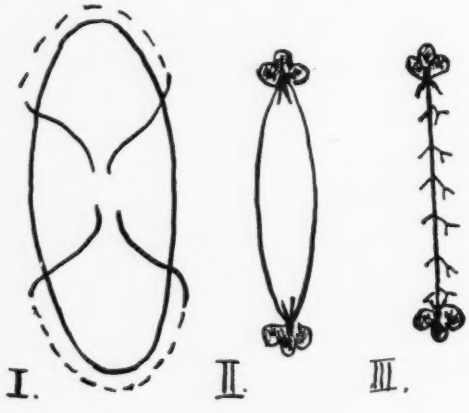

Fig. 4.

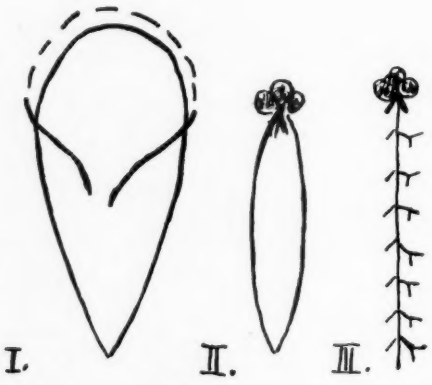

Fig. 5.

Fig. 4.—Elliptic defect. *I*, Putting in the half purse-string sutures; *II*, tying the half purse-string sutures; the defect is now slitlike; *III*, linear union of the transformed defect.

Fig. 5.—Leaflike defect. *I*, Putting in the half purse-string suture; *II*, and *III*, same as *II* and *IV* in Fig. 4.

rarely in others. When the small defect widens to a large one, the tissues bordering it are pushed always farther and farther from the center of the defect (Fig. 3-*I*). The easiest way to close such defects is to bring the tissues together by the same route they used when they were distended, because they are the most relaxed in this direction. Therefore, when closing a circular defect, an attempt should be made to bring the surrounding tissues together in the center of the circle; this is best done by a purse-string suture (Fig. 3-*II, P*). For this reason all defects which are round are closed most easily and with the least amount of tension by purse-string sutures. This cannot be done in cases in which the round defect is very small and in which its surroundings are tough, which is generally the case in epigastric hernias.

There are defects which are neither linear nor round, but elliptic (Fig. 4), egg-shaped or leaflike (Fig. 5). They may be markedly rounded on both ends or relatively long with one rounded and one tapering end. These can be transformed easily into linear ones by making a half purse-string suture on the rounded end or ends (Figs. 4 and 5). Another method to transform rounded, elliptic, egg-shaped, or leaflike defects into linear ones is to exercise the round end or ends. If they are quite small, the margins may be entirely excised so as to permit the lines of incision to meet at acute angles, or a straight incision may be made through them dissecting somewhat the edges of this incision. The latter is advisable in epigastric hernias. In other cases the half purse-string sutures are to be preferred, because they can be done much faster and easier and because tissues which are valuable as plastic material can be saved in this way and also because the closure is generally stronger.

Although the chief purpose of mobilization of the margins of the defect is to bring them into contact, it must often go much farther than this. It must provide that the tissues used for covering the defect should be resistant enough and have vitality enough to produce, when brought into contact, a strong and reliable union of the tissues in the desirable situation.

Now scar tissue cannot be depended upon to do this and therefore must often be thoroughly excised. Atrophic muscles or thin and soft fascial structures are also not suitable for the reliable closure of the hernial defects. Under these circumstances it is frequently necessary to extend the dissection far from the defect to insure the broad contact of reliable tissues.

This is generally possible and in the great majority of cases even easy. Occasionally, however, there are great difficulties, especially in cases in which previous operations have destroyed extensively the tissues surrounding the defect and it is of greater significance when suppurations and necroses follow them. In such cases flaps out of the neighborhood or freely transplanted fascial or aponeurotic grafts must be used for the covering of the defect, or at least for supporting the layers covering it.

IV. WEAK AREAS AND METHODS OF ELIMINATING THEM

There are three kinds of weak areas which threaten the permanency of cure after an operation for hernia: (1) weak areas which always must be left behind in certain kinds of hernias to give room for important anatomical structures, such as the spermatic cord, the femoral vein and artery; (2) defects which are strictly individual, for instance congenital or acquired defects of the external oblique aponeurosis in certain inguinal hernias or conditions, which, although frequent, show great individual variations as, for instance, the weakness or atrophy of the lowest portion of the internal oblique and transverse muscles in

inguinal hernias;[6] (3) the suture lines and the suture perforation canals.

During the course of healing, there is always the possibility that one or more of the stitches may become untied or torn or at least somewhat loosened and, owing to a sudden increase in the intra-abdominal pressure, the whole suture line or part of it may separate. Not only during the healing but even after the formation of the scar, the suture line always presents a weak area, particularly when the edges of the structures are linear, which means that their surfaces of contact are quite narrow. Scars are rigid because they do not contain elastic fibers but only connective tissue; they may withstand pressure, but if they are once stretched by it, as emphasized long ago by Girard, they cannot return to their former size. If this pressure is often repeated, as always occurs in the abdomen because of coughing, sneezing, pressing at defecation, urinating, etc., the scar will be permanently dilated, and with the lapse of time it will become more dilated. It will always become broader and thinner, especially if there is only one scar connecting peritoneum and skin, as occurred, for instance, if all the layers of the abdomen from the peritoneum to the skin were united by

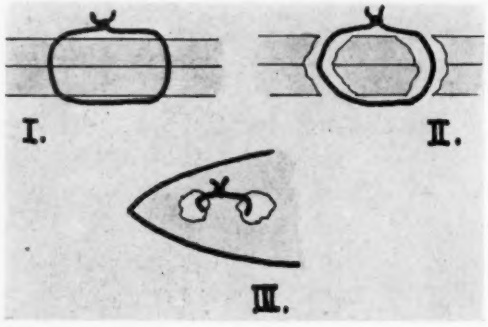

Fig. 6.—How the suture cuts through. *I*, Suture freshly put in; *II*, after cutting through (cross section); *III*, the defects caused by this from above.

through-and-through stitches as was often done a few decades ago, especially after laparotomies in the midline. It may also result when the scars uniting the suture lines of the different layers of the abdominal wall cover each other geometrically; i.e., lie in the same plane; or else when there is a suppuration which makes the open treatment of the abdominal wound going from the skin to the peritoneum necessary.

In all these cases one must reckon with the gradual distention, weakness, and enlargement of the scar which results in hernia or, if the operation is done for hernia, in the recurrence of the hernia.

Not only the suture line itself but also every stitch may cause hernia or the recurrence of the hernia if they connect the preperitoneal and subcutaneous fat. This occurs for example in the Mayo operation for umbilical hernia in which the sutures go through both layers of the aponeurosis covering the defect. Every stitch causes, as already mentioned, an aseptic necrosis, cuts through and produces, in this way,

defects (Fig. 6), which may be filled more or less with scar tissue, which is likely to distend. Moreover, sometimes even before the scar has formed, some properitoneal fat may protrude into the hole which will gradually widen later.

How are these weak spots to be eliminated? (1) By multiplication of the layers, (2) by covering the weak spots with fully reliable resistant tissue, and (3) by carefully avoiding weak areas to be situated so that one lies directly over the other.

If, for instance, the spermatic cord is directed by the shortest route through the abdominal wall, it would pass through all layers at the same vertical plane and this would predispose to recurrence; but, if it is directed through each layer in a different vertical plane, this cause of recurrence is eliminated. Therefore we must separate the weak places and put them as far from each other as possible. This rule must be strictly followed when arranging the suture lines. One must try to put them in a way that no suture line should lie directly below or above the other, that every suture line should be covered with a completely intact layer of tissue, and, if possible also, that the suture lines of the different layers should lie in different directions.

Such a situation of the suture lines diminishes very considerably the dangers presented by the intra-abdominal pressure and especially its sudden increase. It safeguards the suture lines and the scars resulting from them (1) by diminishing the strain to which they are exposed; each suture line protects the other if they are situated as described before. (2) Should some separation of a suture line or some enlargement or weakening of a scar occur, the covering or underlying resistant layer of tissue hinders the development of broader gaps and hinders certainly the protrusion of the underlying structures and in this way eliminates the possibility of recurrence.

V. ADDITIONAL REMARKS

1. No operation starts and ends in the operating room; the preparation and after treatment are of equal importance. After the operation for hernia, the patient should be carefully guarded against any greater increase of abdominal pressure. This is very important for the integrity of the sutures; therefore, one must take care that bronchitis, pneumonia, vomiting, and difficulties in defecation and urination are avoided.

2. The material used for suture must be resistant enough to keep the sutured parts in place at least for one or two weeks.

3. The difficulties of the hernia operation are very different in different cases. There may be difficulties either in eliminating tension or in providing useful tissues for the reliable covering of the defect or in both. Some difficulties may be overcome by favorable anatomical conditions, in such a way that often very large hernial defects can be closed with astonishing facility; whereas, in other cases these diffi-

culties only allow a rather unsatisfactory closure of relatively small defects.

One must never forget that every operation for hernia is a plastic operation and that like other plastic operations the individual peculiarities of the case must always be duly regarded. This is true also for the so-called typical cases but chiefly for recurrent cases and incisional hernias where the normal anatomical conditions are greatly disturbed and often also normal tissues replaced by masses of scar. The more prominent these anatomical changes, the greater is the difficulty of following the rules made for typical cases and the greater the necessity for individualizing.

In the following paragraphs the methods which I have used and which have been used by my assistants, most of them for more than thirty years, are described. They were published in Hungarian, some of them also in German, but they are unknown in America.

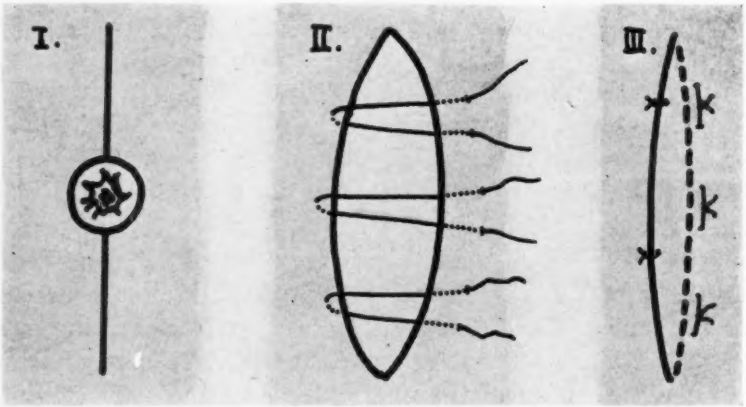

Fig. 7.—Epigastric hernia. *I*, The hole of the linea alba halved by the incision; *II*, mattress stitches; *III*, second row, simple stitches.

VI. EPIGASTRIC HERNIA

Epigastric hernias are generally very small, mostly not larger than a pea or a hazelnut, and the linea alba through which they protrude is generally very rigid; therefore the defect cannot be closed by simple suturing and even less by the duplication of its margins. Besides, the defect is so small that only one single suture could be used and this only at the diameter of the defect which is in the direction of the greatest strain. Therefore it is not secure even if made in the manner of the Lembert sutures as advised by Witzel (Fig. 1).

It is better to transform the small round defect into a long linear one by making an incision 4 to 5 cm. in length in the linea alba, which halves the defect. After retracting the margins of this incision, three sutures are placed, as shown in Fig. 2. It is preferable to use a suture with two curved needles; one needle is inserted through one side of the aponeurotic wound about 1.5 cm. from its margin and then both

needles are inserted through the other edge of the aponeurosis from the inside stitches; one side of the aponeurosis is drawn below the other and now the free side of the above lying one is sutured to the aponeurosis which lies below (Fig. 7-*III*). In this manner the linea alba is duplicated. It is generally sufficient to use three mattress sutures and two superficial simple ones, which are best placed in the intervals between the mattress sutures.

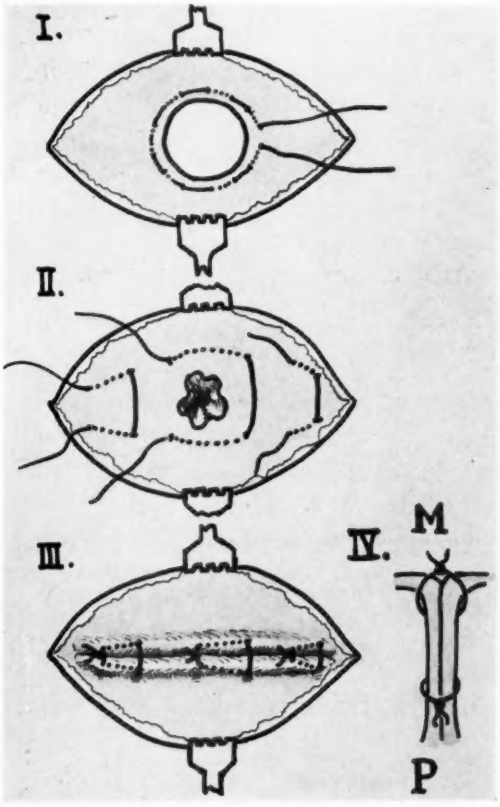

Fig. 8.—Umbilical hernia. *I*, Putting in the purse-string suture; *II*, purse-string suture tied, transverse mattress stitches put in; *III*, mattress stitches tied; *N*, purse-string suture; *P*, tucked in and covered by the mattress suture; *M*, cross section.

This method was first published in 1908[1] and has always given excellent results. Larger epigastric hernias are very rare, but in these the laxity of the surrounding tissues permits their closure with purse-string suture, as will be described for the umbilical hernia.

VII. UMBILICAL HERNIA

After the excision of the sac, the defect of the umbilical hernia is generally round. One must economize with the fascia, which is generally adherent to the peritoneum, and utilize as much of it as possible. After the replacement of the abdominal contents, a strong purse-string suture is installed which goes through fascia and peritoneum; the suture is placed about 2 cm. below and 2 cm. above the tissue formed by aponeurosis and peritoneum and about 1 to 2 cm. from its edge. Some-

what larger bites of tissue may be included in one stitch if the defect is larger and correspondingly less in smaller ones. The beginning of the thread is left long and the end one is brought out at the surface about 2 cm. from the former. The ends of the thread are pulled taut and tied. It is surprising how large openings can be closed with a single purse-string suture (Fig. 8-*I*).

This purse-string suture must be covered, preferably by two or three mattress sutures, which are placed through the aponeurosis so as to bring the soft parts together over it at right angles to the long axis of the body (Fig. 8-*II*), as advised also by Rotter (Fig. 8-*III, IV*). Closure by approximation of the rectus muscle and its aponeurosis after incising the rectus sheath as advised by Maydl is superfluous. I did this in my earlier cases and advised first the purse-string suture as a trick facilitating Maydl's operation,[2] but I discontinued this many years ago and do it only in very exceptional cases. We have obtained very good results with the closure of the defect by purse-string suture and by covering these with simple horizontal folds of the aponeurosis fixed by a few mattress sutures.[3]

VIII. INCISIONAL HERNIAS

In incisional hernias one must try not only to re-establish the normal anatomical structure of the abdominal wall but also to reinforce it as much as possible. The reinforcement of the linea alba can generally be accomplished in quite a typical way; whereas, other cases require a great variety of techniques, depending upon the site and size of the hernia, the kind of incision which was made at the former operation, the amount of scar tissue and the plastic material, etc. The difficulties cannot be realized until the anatomical situation is apparent, and this cannot be determined before the hernial sac, which often overlaps the margins of the defect and may protrude for a considerable distance between skin and aponeurosis, is clearly dissected and excised. The size and form of the defect become apparent only after the replacement of the abdominal contents and the closure of the hernial sac. One can then decide upon the best method of closure.

Median Hernias.—If there is a longitudinal defect which is relatively narrow and tapering at both ends and if the margins are readily mobile or can be made so by undermining, the best method consists of drawing one edge of the defect below the other with mattress sutures and stitching the margin of the overlying side to the aponeurosis which is below. One may use sutures with needles at both ends and place the suture as previously described and as shown in Fig. 7. The two needles are passed through the tissue at a distance 1 to 1.5 cm. apart. The space between the two sutures should be about 1.5 to 2 cm. It is advisable to put in all the sutures before tying them. The tension, which is generally greatest in the middle, is diminished remarkably. After all the mattress sutures are tied and the sides of the defect overlap each other for 1 to 4 cm., the free margin of the overlying side is sutured to the underlying

aponeurosis with interrupted sutures, preferably placing them between the mattress sutures (Fig. 2-*III*). If there is a broad defect with rounded ends, or at least one of the ends is rounded, one must first transform it into a defect with pointed ends by the half purse-string suture (Figs. 4 and 5). This will also simultaneously reduce the length of the gap which can now be united with interrupted sutures (simple or mattress sutures). However, it is generally impossible to produce a broad enough duplication; therefore, it is usually advisable to cover this suture as well as the half purse-string sutures by splitting the rectus

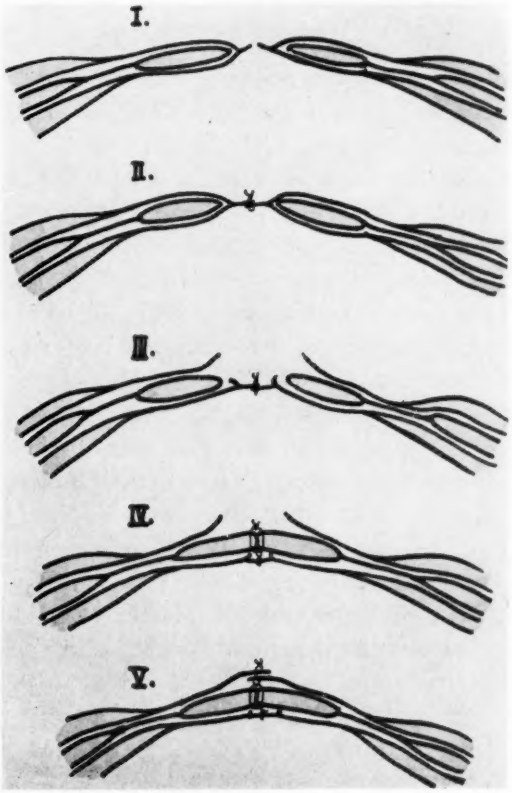

Fig. 9.—Incisional hernia. *I*, Defect of the linea alba; *II*, united; *III*, medial part of anterior rectus sheath opened; *N*, rectus muscles united; *V*, anterior rectus sheath duplicated (cross section). The suture of the medial wound of the rectus sheath (posterior rectus sheath suture) is not illustrated, but it should be done.

sheaths, which were brought close together by the former suture, as near the median line as possible, and sew them together with interrupted mattress sutures. The rectus muscles are then approximated and their anterior sheaths are sutured (Fig. 9). This is not very difficult, especially in multiparous women, and is even easier below the umbilicus. Even in men, defects below the umbilicus can be repaired in most cases relatively easily, but in the epigastric region this often cannot be done as reliably as desired. It is particularly important to approximate the edges of the anterior rectus sheaths because most of these patients are heavy smokers and frequently have bronchitic complications and consequent coughing with resultant tension on the su-

tures. Therefore, in such cases a large flap from the fascia lata covering the rectus muscles and bridging the gap of the rectus sheath is often advisable.

Lateral Hernias.—The great variety of the anatomical conditions in lateral hernias does not allow the description of a typical technique. Nevertheless, certain useful general rules may be observed: (1) Conserve any tissue which may be useful for covering the defect. (2) Expose carefully all normal structures which surround the defect. This generally mobilizes them satisfactorily. If it does not, further mobilization must be done very carefully, sparing aponeuroses, muscles, nerves, and blood vessels as thoroughly as possible. (3) Reconstruct as far as possible the normal anatomical conditions, doubling at least the aponeuroses, especially the aponeurosis of the external obliquus muscle, which is very often conspicuously distended. (4) Make the incision of the rectus sheath near its lateral border; the mobilization of the rectus muscle helps in many cases to close the defects or at least to multiply the covering layers. (5) If the hernia developed in a defect of the rectus muscle itself, so that the hernial defect involves the posterior rectus sheath, its doubling or closure or at least the reduction of the size of the defect and the transformation of its form, by purse-string or half purse-string sutures, may considerably reduce the difficulties of the closure. (6) In cases in which the nerves of the rectus muscle, or at least those of its median portion, have been severed, which occurs in practically all cases in which a longitudinal incision has been made through the rectus muscle or lateral to it, the paralysis of the rectus muscle or at least of its medial part must be assumed; this condition is likely to promote recurrence if the anterior sheath is not reliably reconstructed. Therefore, in such cases the careful union and, if possible, duplication of the anterior rectus sheath or even a fascia lata graft are advisable.

IX. INGUINAL HERNIA

The general principles of the radical closure of hernial defects of the abdominal wall discussed in the first four paragraphs can be realized in the most ideal manner in the overwhelming majority of inguinal hernias, but there are certainly exceptions; namely, some recurrent hernias and some direct hernias.

Recurrent hernias present difficulties chiefly in patients who have been operated upon several times or who have had previous suppuration. As a result of these factors, very valuable plastic material gets lost. For example, the aponeurosis of the external obliquus muscle may become defective or shrunken and the muscles surrounding the inguinal canal may be replaced by hard callous scar tissue. In such extreme cases free grafting of fascia or flaps from the neighborhood* may be necessary, but in most cases one may ensure quite reliable covering by ample mobilization of the rectus muscle. The failing aponeurosis of the external obliquus

*In one of my cases the defect was covered by a flap consisting of skin, fascia lata, and sartorius.[5]

muscle may be replaced sometimes by a broad continuous layer of resistant scar tissue. This is a very incomplete substitution, but better than nothing and although not as valuable is simpler than the graft of fascia lata.

The reliable closure of the hernial defect may be difficult in cases of direct hernias when the internal oblique and transversus muscles are poorly developed. This occurs frequently, especially in people whose entire abdominal musculature is very weak. Not infrequently it occurs in people with good musculature. Defects of the aponeurosis of the external oblique muscle may also hinder the usual closure of the hernial defect; and, if both conditions are present, they may make the reliable covering of the hernia doubtful. But this happens extremely seldom indeed and in most cases the aponeurosis of the external obliquus muscle is so well developed (in large hernias) that it may be largely substituted for the weakened lowest part of the internal oblique and transversal muscles. There is perhaps no other operation in the whole surgical literature which has been as often modified as the widely adopted method of Bassini for inguinal hernia. I too committed this sin thirty-six years ago,[4] but I never had cause to repent it. The method I advised is a combination of the modifications described by Halsted, Berger, and Girard and completed by a few small tricks of my own. It gives excellent results. Many thousands of inguinal hernias were operated upon by this method with practically no recurrences. The very few cases of recurrences which were observed occurred only in cases in which there was suppuration or in which the operation was done for a recurrent hernia.

A broad flap of the aponeurosis of the external obliquus muscle must be prepared with its incision at the upper angle of the subcutaneous opening of the inguinal canal. The incision should be made parallel with the fibers of the aponeurosis and end about 3 to 4 cm. lateral to the abdominal opening. After dissecting the flap, this is drawn downwards and the hernial sac treated adequately. After its replacement the spermatic chord is exposed and if too voluminous some of its veins and the cremaster are resected.

The next step consists of incision of the rectus sheath (Fig. 10-*1*) which may be unnecessary in cases in which the lower part of the internal oblique and transversalis muscles are very well developed. This occurs frequently in congenital hernias of children or young men with strong musculature but very seldom in other cases. In those with poorly developed musculature the rectus sheath must be opened to such an extent that the upper end of the incision should reach the site of the well-developed part of the internal oblique and transverse muscles or slightly beyond it. The length of the incision should be about 5 to 6 cm. in the adult. The rectus muscle and the external margin of the incised rectus sheath as well as the internal oblique and transverse muscles are sutured to Poupart's ligament (Fig. 10-*2*, *3*). Care should be exercised that the suture should include the well-developed part of the latter muscles.

During this stage of the operation, the spermatic chord must be retracted as far laterally as possible and fixed in this position by the most lateral sutures which unite the broad muscles with Poupart's ligament.

The most medial suture should include the whole thickness of Poupart's ligament and the lateral crus of the subcutaneous inguinal opening. Thus for this suture whose knot lies in the deepest layer of the subcutaneous fat I generally use nonabsorbable material (silk);

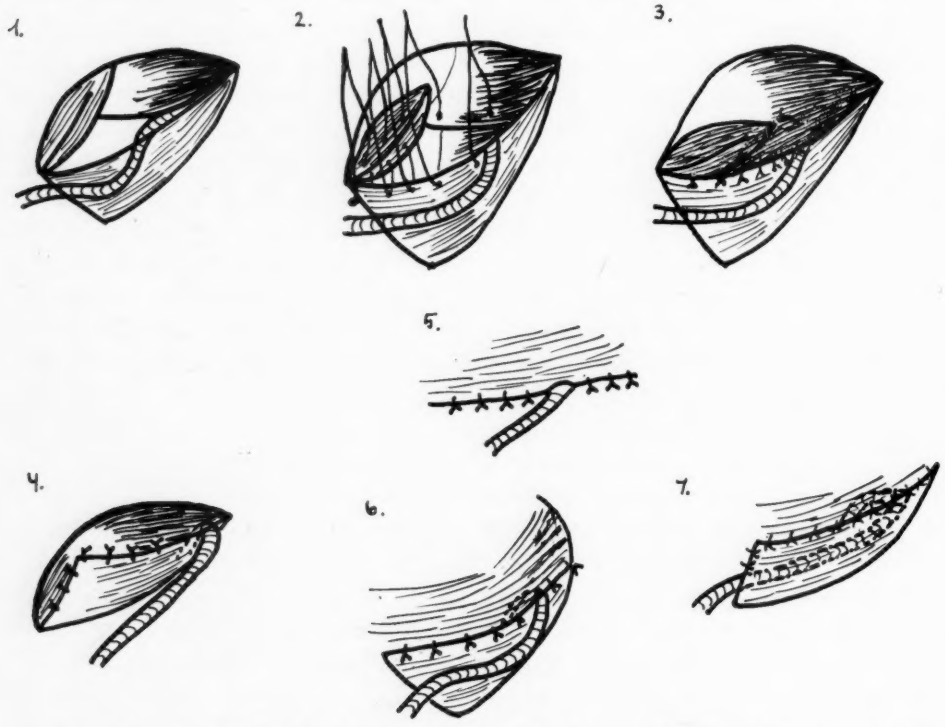

Fig. 10.—Inguinal hernia. *1*, The inguinal defect;[6] lower flap of the aponeurosis of the external oblique put downward, incision of rectus sheath; *2*, deep sutures put in; *3*, tied; *4*, lower aponeurosis flap sutured (mark angulation of the cord); *5*, upper aponeurosis flap pulled down and sutured; *6*, upper aponeurosis flap sewed to Poupart's ligament (mark angulation of the chord); *7*, lower aponeurosis flap sutured to aponeurosis.

whereas, for all the other internal sutures and ligatures, I use catgut. This most medial stitch is exposed to the greatest stress, therefore it is best to tie it last.

The margin of the lower flap of the external oblique aponeurosis is sutured first to the medial margin of the incised rectus sheath (Fig. 10-*4*). The rest of this aponeurosis flap is sewed as far as possible to the internal oblique muscle. The transplantation of the spermatic cord outward is continued (Fig. 10-*4*). When the last suture which lies close to the spermatic cord is tied, the cord is placed on the external surface of the lower flap of the aponeurosis and the upper margin of the incision of the aponeurosis of the external oblique is sewed to Poupart's ligament. The spermatic cord is led out between two

stitches about at the junction of the medial and middle third of Poupart's ligament (Fig. 10-*5*).

By this method the inguinal defect (the weak area between Poupart's ligament, lateral side of the rectus, and lower border of the well-developed parts of the internal oblique and transverse muscles) is covered by three reliable layers (one muscular and two aponeurotic) and the weak areas resulting from the passage of the cord are eliminated. The internal abdominal ring is reliably covered with the above-mentioned three layers; the cord itself passes outward at least 2 to 3 cm.

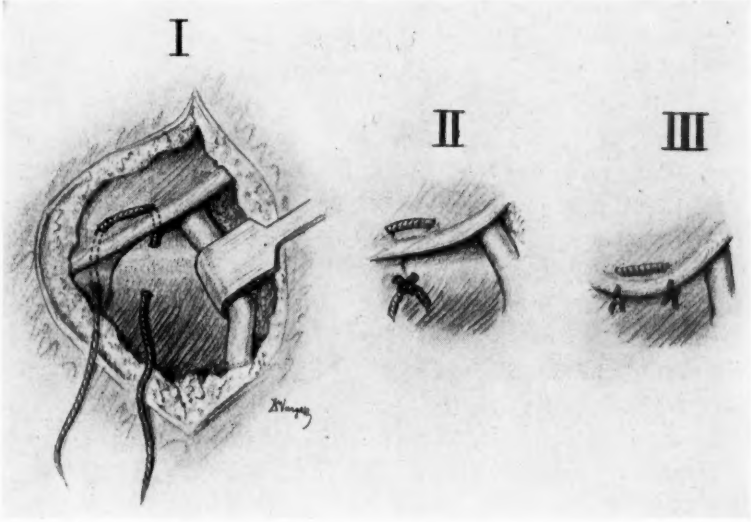

Fig. 11.—Femoral hernia. *I*, Mattress stitch put in through Poupart's and Gimbernat's ligaments and periosteum of the pecten pubis (ligamentum Cooperi); *II*, mattress stitch knotted; *III*, Poupart's ligament sutured to the fascia of the pectineus muscle.

in a narrow musculoaponeurotic canal and, when it reaches the external surface of the lower aponeurotic flap, it is kinked in an acute angle and lies then in a narrow aponeurotic canal and comes out of it through a small transversal opening, between two stitches. Behind it lies a strong aponeurosis.

If the rectus sheath is not opened or even if that is done, the inguinal canal may be closed also (of course, only after the suture of the muscles to Poupart's ligament is completed) by attaching the upper margin of the incised aponeurosis of the external oblique to Poupart's ligament, displacing the spermatic cord as far as possible laterally, and, after kinking it in an acute angle (Fig. 10-*6*), covering it with the lower flap of the aponeurosis (Fig. 10-*F*).

This also gives excellent results because it covers the muscles and the suture uniting the muscles with Poupart's ligament reliably and ensures the angulation of the spermatic cord in a perfect manner. Moreover the most medial part of the suture, where recurrences most frequently occur, is well protected.

X. FEMORAL HERNIAS

The difficulty of the radical operation for femoral hernias is the avoidance of compression of the femoral vein. Therefore, a forced closure of the hernial opening is not only impossible, but even undesirable. If Poupart's ligament is used for the closure and forcibly brought down and attached to the periosteum of the os pubis, one may enlarge the inguinal canal and have an inguinal recurrence. The inguinal route has its own drawbacks (especially when there is a strangulated hernia) and does not exclude with certainty the above-mentioned disadvantages and makes even good partial closure of the crural canal difficult. One must rely on the high ligature of the sac and that is generally not enough. For large crural hernias, especially those that are recurrent, the neighboring muscles may be used. I described, many years ago, the implantation of the sartorius muscle in the crural canal, but I perform this operation only in very exceptional cases. The splitting of the rectus sheath and uniting of the rectus with the periost of the os pubis and the covering of this with a flap of fascia lata is easier and ensures pretty good closure. But in small hernias even this is too much, so I generally put a mattress suture 1 to 1.5 cm. over Poupart's ligament in the aponeurosis of the external oblique. The medial part of this suture also passes through Gimbernat's ligament (Fig. 11-*I*) and then both threads go through the periosteum of the os pubis. The lateral suture is placed as near the femoral vein as possible without compressing it. After this mattress suture is tied, Poupart's ligament is sewed with a few stitches to the fascia of the pectineus muscle (Fig. 11-*III*). This is somewhat stronger than the similar method of Kocher, Payr, etc., but there is really no absolutely reliable method for curing femoral hernia and one must in these cases be more prepared for recurrences than in any other form of hernia, except those incisional hernias which lie in the median line above the umbilicus, especially when the patients are men and the hernias large.

Ultra posse nemo tenatur. One cannot cure everything, but one must try to do one's best, which means in hernias, multiply the layers and cover the weak areas as reliably as possible under the given anatomical conditions.

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OSTEOMYELITIS OF THE JAWS*

An Analysis of Fifty-Nine Patients

Earl C. Padgett, M.D., Kansas City, Mo.

(From the Surgical Department of the University of Kansas School of Medicine and the Kansas City Western Dental College)

THE influence of the teeth and tooth buds on the course of infection of the jaw bones in contrast with the other bones of the body lends a unique interest to osteomyelitis of the jaws. The danger of initiating a severe infection of the jaws, when he uses bad judgment in regard to the optimum time to inflict operative trauma, is a threat which often hangs over the head of the exodontist. In this paper I wish to discuss generally what one might term "frank" osteomyelitis from the standpoint of my own material during the past ten years. In the group are fifty-nine patients. The cases of osteomyelitis following fracture have been excluded from the group. As a matter of fact, in both the acute and chronic alveolar abscess an osteomyelitis is present, but it is not common practice to use the term osteomyelitis in describing these lesions.

In the group are two unique examples of a type of slowly progressive osteomyelitis of the upper jaw which extended to the skull, producing death eventually because of a meningitis. I wish to suggest that these two patients are possibly examples of a more or less unique clinical entity on which little stress has been placed previously.

The maxim to be drawn from a study of the pyogenic group as a whole is that, as a rule, treatment during the acute stage should be conservative, but on the contrary, after sequestration has been effected, fairly thorough and somewhat radical treatment is the most effective.

INCIDENCE

Frank osteomyelitis of the jaw bones is not very common. Leibold considers it eighth in frequency. The femur, tibia, humerus, radius, ulna, vertebrae, and os calcis precede the mandible in the order named. Wilensky in the Mt. Sinai series (1924-1930) found 450 patients with acute and chronic osteomyelitis of all kinds. The upper and lower jaws were involved in 39 instances.

In my series of 59 patients, 22 were below the age of 16 years and 37 were older; 21 cases occurred in the female and 38 in the male sex. The mandible alone was affected 38 times and the maxilla alone 16 times and in 3 patients both the mandible and the maxilla were involved. One of these was an osteomyelitis due to excessive irradiation and the other two were of the pyogenic type. In 2 other patients with a slowly progressive osteomyelitis of the maxilla the inflammatory

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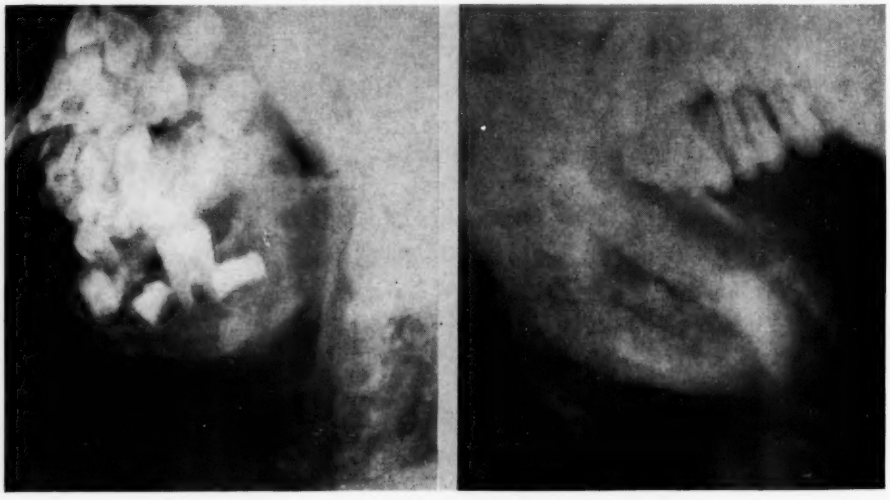
process also eventually involved the base of the skull and the frontal bones, although originally the process started in the maxilla.

The etiology is interesting. The disease in over one-third of the patients (25) was associated with a peridental infection. In 11 patients the disease was associated with an extraction during the acute stage of a pulpitis or a peridental infection (Fig. 1*A*); 12 developed from an infection of the pulp cavity unassociated with extraction and in 2 patients too much mercury was the basis of the necrosis.



A. *B.*

Fig. 1.—*A*, This boy had a toothache August, 1933. The jaw became tender and he had some fever. The tooth was removed. He became very ill. A good many of the lower teeth became loose and dropped out. When I saw him Nov. 5, 1933, his jaw was underdeveloped. Most of the teeth and the tooth buds were absent. Here and there was some dead bone all through the mandible. The mandible was explored; all infected tooth buds were removed; all granulating sinuses were laid open; and all overhanging bone was removed. After this operation the boy was cured, but the lower jaw has not developed as much as it should.

B, Roentgenogram of boy aged 16 years. He had a blister on his foot and two days later developed an osteomyelitis of the lower jaw. He was seen by me three months later. At that time he had lost all of his teeth and tooth buds and the alveolar ridges of the lower jaw. There had been considerable new bone formation and after a moderate sequestrectomy the jaw healed. Some months later the gingivolabial sulcus was deepened and a denture fitted.

In 5 patients trauma to the jaw in the region of the tooth initiated the infection. In 4 the bone became infected after an operative extraction. In 2 of these individuals, although the tooth or root was removed, it was found that the dentist had fractured the jaw in removal. In 1 of these patients the jaw was very atrophic. In 1 patient a corrosive fluid had been injected.

In 29 patients the origin of the lesion seemed to be other than a peridental infection or trauma. Of these, 4 were of the blood-borne type (Fig. 1*B*); 19 were due to syphilis; 6, to excessive irradiations; 1, to noma; 1, to trauma of the cheek; 3, to leucemia; and 3 possibly originated from an infection of the maxillary sinus.

If one compares the etiology of these cases with certain other published groups, for example those of Blair, one gains the impression that

possibly the dentists at the present time do not so often extract a tooth during the acute stage. In Blair's 39 cases, published in 1923 and 1926, wherein he emphasized particularly the dangers of extracting the ulcerated tooth during an acute stage, 31 were associated with extraction of a tooth during the acute stage. The number of cases (9) due to syphilis in our series is rather high. Most of these individuals were referred to me because a reconstruction operation would be necessary eventually. Osteomyelitis due to excessive irradiation in the older series of published cases is not listed. However, today it is not uncommon to have an extensive osteomyelitis due to a secondarily infected bone irritated by excessive irradiation. Although no cases of slight adjacent necrosis purely due to the local effect of the implantation of radium were included, in the series were found 6 cases with necrosis of the complete thickness of the mandible after excessive irradiation.

ORGANISMS

Rosenow, Gilmer, and Moody and Haden present evidence to show that the common organism associated with a peridental infection is the *Streptococcus hemolyticus*. Less commonly the staphylococcus or a mixed infection causes the infection. When osteomyelitis of the jaws is caused by a blood-borne organism, the offender is usually the *Staphylococcus aureus*. A blood-borne infection, however, occurs only rarely. When trauma or disease is the cause of a secondary infection of the jaw bone, a mixed group of organisms of the variety commonly found in the mouth usually is present.

PATHOLOGY

The pathology of an osteomyelitis of the lower jaw is influenced especially by two factors: (1) the presence of dental elements in the bone and (2) a unique blood supply. The pathology of the upper jaw may be influenced particularly by four factors: (1) the presence of the dental elements, (2) a unique blood supply, (3) the fact that the bone is of membranous origin, and (4) the presence of the maxillary sinus.

Besides the preceding special factors, an osteomyelitis of the jaws shows the same pathologic changes as any other fibroblastic tissue containing a deposition of lime salts in the ground substance. Briefly, as the products of inflammation accumulate under tension, the vessels within the bony spaces become thrombosed. The bony trabeculae tend to be absorbed and the products of inflammation following the path of least resistance seek an outlet through the bony cortex. After the cortex is perforated, some elevation of the periosteum occurs before it, in turn, is perforated. The cellular reaction encountered as a sequestration takes place and involucrum is formed in the jaw bones is, on the whole, the same as that which occurs in other bones and needs little comment here, save to emphasize that a line of demarcation is formed between the live and dead bone which is usually complete in from two to three months

and that as long as any dead bone is present the opening in the periosteum will persist through which pus and debris will drain. It is to be remembered that the maxilla is a membranous bone and therefore it lays down little or no new bone. The products of inflammation about a tooth cause the fibrillar connections between the peridental membrane and the cementum of the tooth to be disrupted. The cementum once denuded remains denuded. The cementum has no blood supply and does not exfoliate. As long as the tooth is present, the uncovered cementum acts as a nonviable, irritating foreign body. Before the teeth are erupted the dental sacs may be involved in the inflammatory process; when this occurs, the unerupted tooth or teeth become isolated, nonviable, and act as foreign bodies in a manner similar to a sequestrum.

The amount of devascularization of the bone depends upon the degree of stripping of the periosteum on the one hand and the degree of central thrombosis on the other. Thus, if sufficient periosteum is stripped from either bone of the jaw while coincidentally the central blood supply is blocked, complete necrosis of the whole bone may ensue. This is especially true of the mandible where the centrally located inferior dental artery furnishes blood to the central part of the bone.

The blood supply of the upper jaw is also somewhat unique. Wilensky recently has emphasized that the upper jaw receives the blood supply principally from two arterial branches, the anterior dental which passes in front of and the posterior dental which passes behind the antrum to anastomose with each other in the superior alveolus. As the blood supplied by the periosteum to the maxilla is quite meager, the size of the sequestration is particularly influenced by the location of the thrombus and the amount of thrombosis that may be present. In the type of osteomyelitis that may occasionally occur in nurslings, this may be particularly true.

CLINICAL COURSE

The outstanding early local symptoms of an osteomyelitis of the jaw, as in osteomyelitis elsewhere in the body, are severe, aching, throbbing, deep-seated pain, or in the recumbent position a developing local tenderness, worse on percussion, and local swelling which eventually fluctuates if the tension within is not too great. The general symptoms are those which accompany any pyogenic infection. As a rule as soon as the periosteum is perforated the pain decreases, but, if the pus is evacuated in the soft tissues about the bone, the swelling tends to increase rapidly. The location of the main infection and the direction the products of inflammation take as they enter the soft tissues influence the symptoms considerably. In involvements of the lower jaw the pus sometimes extends into the deep tissues of the submaxillary region, the upper deep cervical region, or into the tissues into the region of the anterior pillars. Involvement of the muscles of mastication

will cause trismus and swallowing will be impeded when the tissues of the floor of the mouth or pharynx are invaded. In osteomyelitis of the upper jaw the inflammatory products may extend into the tissues of the anterior cheek, nose, or palate. When the tissues of the hard palate are undermined, there may be no particular redness because of the compact structure of the soft tissues in this region.

When drainage is provided either spontaneously or by incision, the pain, swelling, and constitutional reaction largely subside, but sinuses with pouting projections of granulation tissue persist as long as dead bone or dead tooth structure remains in situ. A few cases of osteomyelitis of jaw bone result in slow bone absorption without actual gross death of bone, but as a rule a definite sequestrum is formed, which takes about two or three months to become separated from the live bone.

Often age influences the course of osteomyelitis of the jaw considerably. In nurslings the characteristic invading organism is one of great virulency and as a rule a septicemia either precedes, accompanies, or follows the infection. In children often the damage to the tooth buds which necessitates their removal may seriously interfere with the subsequent growth of the jaw bone even if the amount of sequestration does not cause serious deformities.

In the bone necrosis seen accompanying a primary blood disease, the soft tissues about the necrotic bone usually show only a slight reaction. The constitutional symptoms tend to be more severe than the local condition would lead one to suspect. The general appearance of an adult, rather pale and yellow, and the fact that no other obvious etiology seems to be present suggests to one immediately that a blood smear is advisable.

The outstanding feature of an irradiation necrosis is the chronicity of the course, the continued pain, the slowness of sequestration, lack of tendency for either the bone or the surrounding soft tissue to show any of the ordinary tendencies of normal tissue toward healing, and finally lack of resistance to secondary infection.

When a child is seen with evidence of a bone infection in the region of the epiphyseal cartilage of the condyle, rather often it is difficult to be certain whether or not one is dealing with a purulent arthritis of the temporomandibular joint or a true blood-borne osteomyelitis of the type more commonly seen in the other long bones. The region in front of the ear is swollen and tender and in either case great difficulty is experienced in moving the lower jaw. The joint cannot be aspirated well. The only possible distinguishing signs are that the swelling and point of maximum tenderness should be lower in an osteomyelitis and less difficulty should be experienced in moving the lower jaw.

LOCATION AND SIZE OF SEQUESTRUM

Classification as to the location and the size of the sequestration is of some interest. This in our series was as follows: In the body of the

mandible alone 6 showed a small sequestrum, 13 a sequestrum of medium size, and 12 a large sequestrum but not involving a whole section of the bone. In 6 others the sequestrum was so large that the continuity of the bone was discontinued. In 1 the sequestrum was the ramus and in 1 the condyle and the neck. In the maxilla alone 2 showed a small amount of sequestration, 11 a medium amount, and 5 a large amount. In 3 patients sequestrum appeared in both mandible and maxilla. In 2, 1 following mercury and 1 following excessive irradiation, the sequestrum was of large amount. Characteristically in the necrosis due to mercury the alveolar ridges were lost principally. In irradiation necrosis characteristically the whole thickness of the mandible becomes secondarily infected and necrotic. In the syphilitic cases the palatal process or the frontal process of the maxilla was involved most commonly. In the latter case the bone usually was involved by extension from the nasal bones.

THE ROENTGENOGRAM

Early some mottling of the bone usually is to be seen in the roentgenogram. Usually after six or seven weeks one may get some idea of the probable amount of sequestration. A hazy, moth-eaten, uneven outline between the edges of the live bone and the dead bone begins to become evident and the sequestrum shows as an irregularly shaped piece of bone lying in a cavity surrounded by either uninvolved bone or new bone. Later the sequestrum loses some of its density. In those cases in which the necrosis is the full thickness of the jaw, the roentgenogram aids in judging the extent to which the involucrum or new bone has formed. Repeated roentgenograms over a period of six to nine months may be necessary before there seems to be sufficient new bone not to jeopardize the contour of the lower face when the sequestrum is removed.

TWO PATIENTS WITH UNIQUE PROGRESSIVE OSTEOMYELITIC AND SOFT TISSUE DESTRUCTION OF UPPER JAW REGION, BASE OF SKULL, AND FRONTAL REGION*

Of unique interest are the patients with a progressive osteomyelitic and soft tissue destructive process of the upper jaw region, frontal region, and the base of the skull. In the literature an exact description of a similar clinical course has not been found. The clinical courses of these two patients were strikingly similar. The patients were both men in middle life. Previously they had been in good health. In each case the onset seemed to have been associated with antral infection and trauma (Figs. 2, 3, and 4). Accompanying and following the

*These two patients were seen in consultation. The first was a patient of Paul Stookey (Figs. 3 and 4) and the second a patient of Charles Dennie (Fig. 2), of Kansas City. It is with their consent that I publish them. The comments are mine. Stookey finally concluded that his case might be a case of leishmaniasis and Dennie finally concluded that his case might be one of malignant syphilis. I have placed a somewhat different interpretation on them.

involvement of the maxillary bone, a small ulceration developed in the soft tissues anterior to the maxilla. Sloughing of the soft tissues and necrosis of the bone continued slowly. Both small and large sequestra formed. First, the front of the face and the nose sloughed away. Finally the invasion extended to the frontal bone and the base of the skull. In both patients the course of the disease was known to have been in the neighborhood of eighteen months before death from a meningitis.

One of the patients (Fig. 2) had a history of receiving some "shots" several years previously. The Wassermann test of each was negative. Each received intensive antisyphilitic treatment. Each had a history of antral insult, one a kick by a horse (Figs. 3 and 4) and one an antral operation. Cultures, both aerobic and anaerobic, were run on both.

A. *B.*

Fig. 2.—Case of progressive osteomyelitis of the maxilla which started after traumatic injury to the antrum successively involving the frontal bones and base of skull, death finally resulting from a meningitis. *A*, Photograph taken Jan. 13, 1939; *B*, photograph taken Aug. 18, 1939, just before his death. (Case of Dennie.)

Biopsy specimens were taken on both and finally autopsy was performed. Each had tests for glanders leishmaniasis (N.N.N. medium and tartar emetic). The biopsy reports were consistently granulation tissue. It is known, however, that in gangrenous conditions about the face and mouth spirochetes are usually present. From one of these some spirochetes were seen, but the pathologist (H. R. Wahl) was unable to identify them as the *Spirochaeta pallida*. They were probably secondary invaders. Guinea pig inoculations were made without positive findings. One patient (Figs. 3 and 4) had been in Mexico. The

other patient (Fig. 2) had never been out of this country. In one case (Fig. 2) rabbits were inoculated with the sera of some of the involved tissue.

A. *B.*

Fig. 3.—Second case of progressive osteomyelitis of the maxilla. The first indication of the onset of the condition followed an injury to the antrum. The osteomyelitis successively involved the frontal bones and base of skull and resulted in patient's death from a meningitis. *A*, Photograph taken Sept. 22, 1931; *B*, photograph taken Sept. 15, 1932.

A. *B.*

Fig. 4.—*A*, Same patient as shown in Fig. 3. Photograph taken Sept. 15, 1932; *B*, photograph taken Dec. 7, 1932, at the time of his death. (Case of Stookey.)

Sometimes a pyogenic osteomyelitis develops in the frontal bone following a frontal sinusitis which progressively involves the bones of the

dome of the skull. Blair has reported a few such cases. It has occurred to me that these cases may be of a similar nature. Both the maxilla and the bones of the dome of the skull are membranous bones. They have little or no ability to lay down new bone and there is possibly less tendency to limitation of a low grade inflammatory process than would be the case in nonmembranous bone.

PROPHYLAXIS

A question of great importance is whether or not one should extract a tooth during the acute stage of peridental infection. Exodontists of wide experience state that in most instances early extraction of the tooth at fault will give drainage and may prevent the onset of a true osteomyelitic process. They believe that those few cases that go on to show the signs eventually of an osteomyelitic process would have done so anyway. On the other hand, surgeons who see the frank cases of osteomyelitis have been inclined to point out the dangers of extracting a tooth during the acute stage.

Probably in addition to the factors of virulency of the infection and the resistance of the patient the crux of the situation is the amount of trauma inflicted in the removal of the tooth. If the tooth can be simply and easily lifted out of its socket, it is probable that the operation is similar to the one of early trephining in an osteomyelitis of a long bone. If the infection does not have clinical signs of great virulency, early extraction with little trauma will give relief from pain and usually is generally beneficial. On the other hand, if considerable trauma is inflicted in the removal of the tooth, it would be somewhat similar to doing a radical procedure early in the course of an osteomyelitis of a long bone, a procedure long known to be distinctly contraindicated.

It probably also should be remembered that the analogy in comparison with osteomyelitis of the long bones in general breaks down somewhat so far as the organism that most often causes an osteomyelitis of the jaws is concerned. An acute peridental infection is usually caused by *Streptococcus hemolyticus*, while the organism encountered elsewhere is as a rule *Staphylococcus aureus*. This fact warns one to lean to the conservative side when in doubt.

O'Kell and Elliott ran blood cultures on a series of patients following the extraction of teeth for septic mouths and they found that "within a few moments after the extraction of teeth from obviously septic mouths, a transient streptococcal bacteremia lasting a few minutes may be observed in 75 per cent of cases." The observation is of great importance to the subject in hand.

From a perusal of the cases that I have seen, I would judge that one might use the same type of surgical judgment in regard to this matter that should be used in regard to an acute, subacute, or chronic osteo-

myelitis in bones other than the jaws. If the infection does not give the clinical signs of great virulency, if the general health of the patient is good and little trauma is necessary to extract the tooth, I should judge that early extraction may be beneficial. However, if the clinical signs of the infection suggest considerable virulency, if the patient is debilitated and it appears that the extraction would be associated with considerable trauma, early extraction should not be performed.

TREATMENT OF THE OSTEOMYELITIS

After development of a true osteomyelitis, the patients reported herein have been treated conservatively while the process was acute. As soon as definite evidence of localization of the products of inflammation occurred, the soft tissues were incised, but operation on the bone was considered contraindicated. Whenever internal incision seemed unlikely to give adequate drainage, drainage by external submandibular incision was made.

During the period between drainage of pus and sequestration, the treatment was symptomatic and supportive. When the time interval was sufficient for the sequestration to have separated, an approach was made through an incision in an inconspicuous position which allowed fairly direct approach. The sinus tract was followed; all dead bone was removed. If a sinus tract led to a tooth root or to a tooth bud, it was removed. All overhanging edges of the bone were removed. Thus, one side of the bone was saucered out so that the soft tissue could fall into the defect. For the first week the wound was gently packed open with a strip of iodoform gauze which as a rule was changed every other day.

The exception to the above procedure as to time of the sequestrectomy occurred in two patients with gross sequestration of the whole thickness of the lower jaw; in one it was deemed wise to wait six months and in a second nine months to allow sufficient new bone to form to maintain the contour of the lower face. In the majority of cases only one operation was found to be necessary to effect a cure if the operation in the chronic stage was rather thoroughly and completely performed.

NUMBER OF OPERATIONS AND PROGNOSIS

In our series the two patients with the progressive osteomyelitis of the upper jaw, base of the skull, and frontal bone, the patient with noma, the 3 patients with leucemia, and 2 of the patients with irradiation necrosis died without operation. It was judged that either the patient's general condition did not warrant any operative procedure or that the disease was of a type not amenable to surgery. Thirty-four of the pure pyogenic osteomyelitis cases were cured after one operation; 3 had more than one sequestrectomy. None of them died. In 2 cases of irradiation necrosis one-half of the mandible was resected be-

fore separation was complete for the purpose of relieving pain. In the 2 others nothing was done unless separation of the sequestrum was complete. In most instances the sequestrum in the syphilitic cases finally separated and was exfoliated spontaneously or was removed by forceps without anesthesia.

SUMMARY

Although frank osteomyelitis of the jaw bones is not very common, the disease, nevertheless, is of considerable seriousness and often does not receive the care most likely to effect the best results. In children, due to interference with the nonerupted teeth, the resulting deformity of growth is likely to be considerable. In both children and adults gross loss of a part of the mandible, especially, may produce a deformity and considerable disability.

Two patients who suffered from a unique chronic progressive osteomyelitis of the maxilla, base of the skull, and frontal bones which terminated in a fatal meningitis after a period of about eighteen months are described. It is suggestive that the process seemed to start in the maxillary sinus and progressively involved membranous bone.

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PARAVERTEBRAL PROCAINE BLOCK IN THE TREATMENT OF POSTOPERATIVE ATELECTASIS

A Preliminary Report

John A. Gius, M.D., Med. Sc.D., Portland, Ore.

(From the Department of Surgery, University of Oregon Medical School)

WITHIN recent years the importance of atelectasis as the predominant early postoperative pulmonary complication and the primary condition in the development of postoperative pneumonia has been widely recognized. In a review of the literature published elsewhere[1] the different aspects of the problem, including pathogenesis and treatment, have been discussed.

Many authors (Head,[2] Powers,[3] Patey,[4] Carlson,[5] Beecher,[6] Capelle[7]) agree that pulmonary hypoventilation occurs after nearly all abdominal operations. This appears to be the result of the incision placed in the abdominal wall which directly and reflexly interferes with abdominal and diaphragmatic excursion. That this effect is particularly marked after upper abdominal operations is in agreement with the clinical observation that incidence of pulmonary complications is greatest after operations on the stomach and duodenum, biliary tract, pancreas, and spleen. Most of this impairment of ventilation can be ascribed to wound pain producing voluntary inhibition and reflex spasm of the abdominal muscles as well as the diaphragm itself.[7] That the diaphragm following operation is high and relatively immobile has been repeatedly observed. The frequency with which transient physical signs can be demonstrated in the right lower lobe is another manifestation of this impairment of function. Capelle[7] has shown that continuous anesthesia of the abdominal wound will result in reactivation of the diaphragm as well as a return of the vital capacity from the usual 20 to 40 per cent immediately after operation to 90 per cent of normal.

In addition to hypoventilation another significant predisposing factor exists, the loss of the ability to cough effectively following operation. Secretions which are normally removed by cough, ciliary action of the bronchial epithelium, and peristaltic activity of the bronchioles tend to thicken and adhere to the bronchial walls and thus may be removed incompletely or only with great difficulty. If the patient is unable or unwilling to cough, the mucus may remain and produce lobular or even lobar obstruction. Absorption of the air entrapped distally soon occurs and atelectasis results. Unless vigorous measures are promptly undertaken to remove the obstructing mucus, organisms usually derived from the upper respiratory passages invade the collapsed area and produce the much more serious complication, pneumonia.

The most effective measures for securing evacuation of the plug and re-expansion of the collapsed area are: active hyperventilation and cough, either voluntarily or by means of carbon dioxide inhalations; fre-

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quent changes in position and postural drainage; vigorous percussion over the collapsed lobe; expectorants to thin the retained secretions (ammonium chloride or potassium iodide); and removal of the secretions by intratracheal suction or bronchoscopy.

It is a common observation that most patients cannot cough effectively after operation because of wound pain. If cough is present, it is most frequently of the weak, restrained, unproductive type, though it is obvious that tracheobronchial secretions necessitating removal are present. Morphine has been advised to relieve pain and secure more effective coughing, but this drug decreases the sensitivity of the cough reflex and depresses respiration in general; therefore, it has marked disadvantages. Capelle[7] has shown that an increase in the vital capacity amounting to only 3 to 5 per cent follows the administration of morphine. Thus the amount of air which can be utilized in sweeping the bronchial tree free of its contained mucus is not appreciably augmented.

It may be reasoned that relief of abdominal wound pain possibly may cause restrained coughing. It is, however, unlikely that this mechanism could work in this direction, since cough results from stimulation of the respiratory mucosa by the presence of foreign material in the air passages. Thus, pain in the wound could logically serve only to inhibit the action of the muscles of respiration or more specifically the abdominal muscles which are so necessary for forcible and effective coughing.

Because of these facts I have reasoned that elimination of wound pain by means of paravertebral novocain block would produce an increased pulmonary ventilation and more effective cough reflex. If this method could be used both prophylactically and therapeutically in atelectasis it seemed that this would be a particularly promising procedure with which to combat postoperative atelectasis.

Several preliminary observations were carried out at Presbyterian Hospital, New York City, in an attempt to measure the relative changes in thoracic and abdominal excursion in postoperative cases after paravertebral block. These experiments showed some augmentation in the diaphragmatic component. In an attempt to secure prolonged anesthesia a local anesthesia in oil (neothesol) commonly used in anorectal surgery was tried in a few cases without conclusive results. This work is to be continued.

In the meantime, however, two cases* of atelectasis following appendectomy were encountered in children. Such remarkable results followed the induction of paravertebral block anesthesia of the wound that I believe this method to be of sufficient value to report at this time.

CASE 1.—R. A., male, aged 7 years. The patient was admitted to Doernbecher Hospital for Children on Jan. 19, 1940, with a history of diarrhea of three days'

*Since this paper was submitted a third case presenting essentially the same picture was treated in a similar manner with equally satisfactory results.

duration. On Jan. 18 he developed lower abdominal pain and vomiting. Castor oil had been administered on the day of onset of symptoms. During the preceding week the patient had been convalescing from the "flu" and during this time had had some nasal discharge, cough, malaise, and anorexia.

The essential physical findings consisted of a temperature of 100.8° F.; pulse, 88; respiration, 22. The nose and pharynx were definitely injected. The tonsils were large and infected. Heart and lungs were negative. Moderate tenderness and spasm were found in the right lower quadrant. Urinalysis was negative except for acetone +++, diacetic +. White blood count, 25,000; polymorphonuclear leucocytes, 79; small lymphocytes, 13; monocytes, 4; sedimentation rate, 9 first 15 minutes, 43 in 45 minutes. *Clinical Diagnosis:* Acute appendicitis, subsiding influenza.

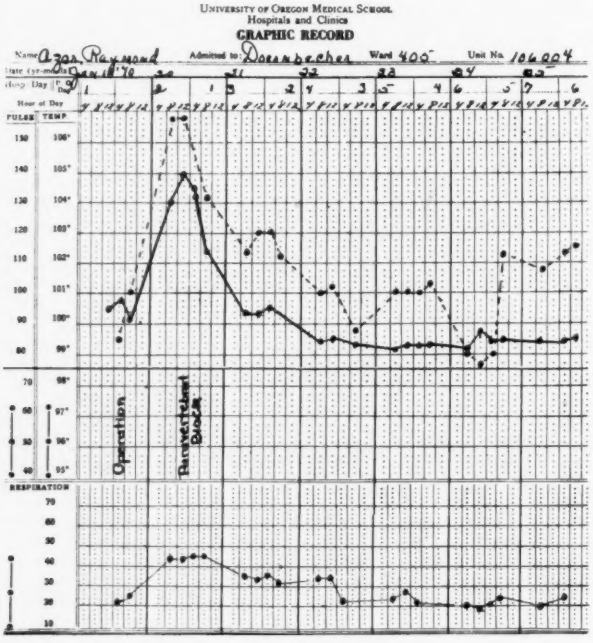


Fig. 1.

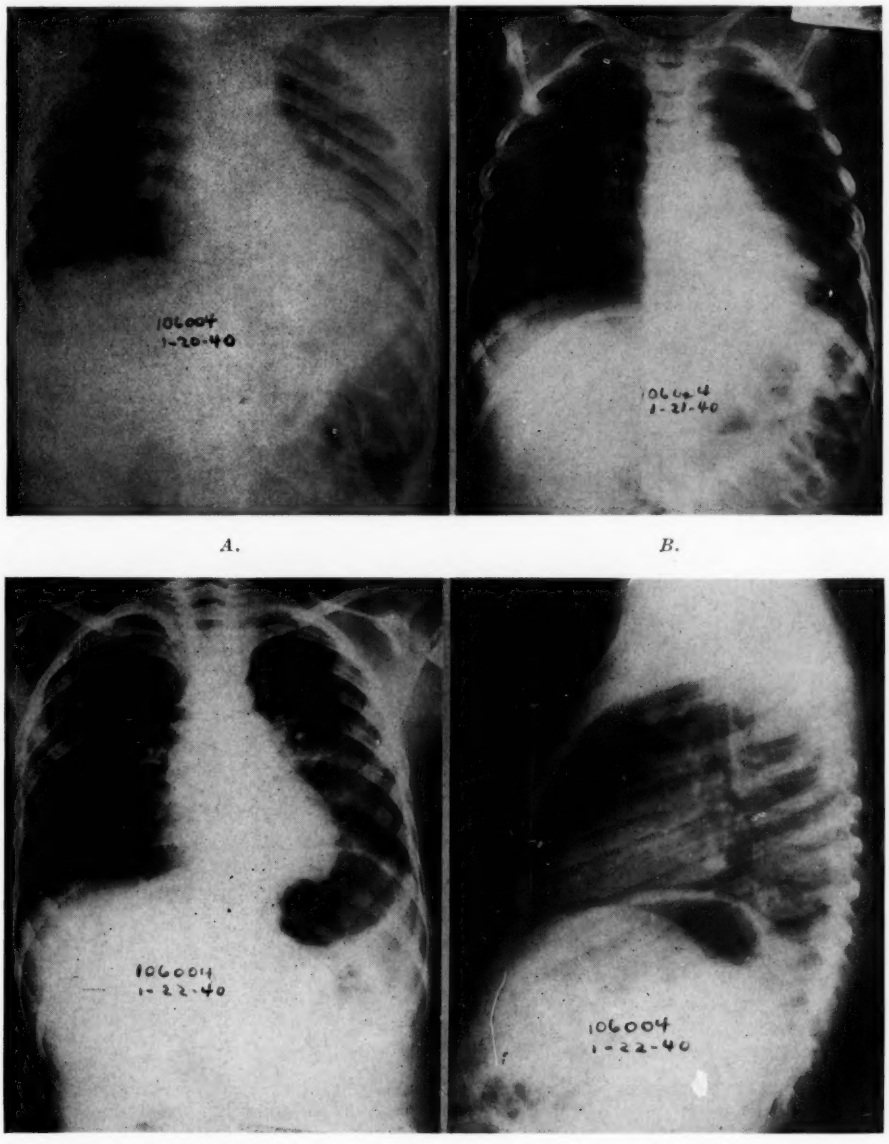
An infusion of 750 c.c. 5 per cent glucose in saline was given and appendectomy was performed through a McBurney muscle-splitting incision under open-drop ether anesthesia. A large, acutely inflamed appendix with a small abscess was found. Cultures of the abscess later showed gram-negative rods, resembling *B. influenzae* and a few hemolytic streptococci. A Penrose drain was placed down to the peritoneum and the wound was closed loosely.

Postoperatively the patient was given parenteral fluids and codeine. On the first day after operation the temperature spiked to 105° F. and the pulse and respiration rose to 160 and 44 respectively (Fig. 1). It was noted that the patient was trying to inhibit cough which seemed dry and unproductive. Attempts to get him to cough were of no avail on account of pain in the wound.

Examination of the chest revealed a slight lag on the left. Some suppression of breath tones and dullness were noted over both lower lobes posteriorly near the spine. The breath tones had a bronchial quality in the region of the left hilum. There was no demonstrable mediastinal shift.

X-ray of chest showed some increased density of the left base with narrowing of the intercostal spaces, no mediastinal shift. Impression: Atelectasis left lower lobe.

Paravertebral block was done (Dr. V. Larson) with 1 per cent procaine with cobefrin solution. Four cubic centimeters were deposited in each of the following spaces: dorsal 10, 11, 12, and lumbar 1 and 2. Immediately following the block the patient began to cough effectively and without pain in the wound. He was



A. *B.*

C. *D.*

Fig. 2.—Case 1. *A*, (Jan. 20, 1940) There is a suggestion of an increased density at the left base behind the heart. There is definite narrowing of the fourth to eighth rib interspaces. *B*, (Jan. 21, 1940) The film shows a definite density behind the heart on the left, as well as narrowed rib interspaces. *C*, *D*, (Jan. 22, 1940) There is definite diminution in the density at the right base. The diaphragm is elevated because of gas in the stomach. A film taken on Jan. 24, 1940, showed the lung fields to be clear.

placed in the lateral position with the foot of the bed elevated, turned frequently, given carbon dioxide inhalation every hour, and ammonium chloride 10 grains every four hours.

He continued to cough voluntarily during the remainder of the day, and small amounts of mucus were expectorated. By evening the temperature had fallen to

102.2° F., and it was found that the lung fields had cleared somewhat. However, there was still some suppression of the breath tones and impaired resonance at the left base. No râles were heard.

On the following day the temperature was 100.4° F.; pulse, 120; respirations, 32. Cough was vigorous but not very productive. The chest signs were somewhat less marked than previously, but some suppression of breath tones persisted. These cleared on the following day and except for slight drainage from the wound the remainder of the convalescence was uneventful.

CASE 11.—(No. 37012.) C. D., female, aged 11 years, was admitted to Doernbecher Hospital for Children on Dec. 8, 1939, for pain in the lower abdomen of one day's duration. The patient had had a cold for a week which was apparently clearing satisfactorily. On the night before admission she had complained of epigastric pains which were later referred to the right lower quadrant. There had been no nausea or vomiting. Some constipation had been noted previously, but no cathartics had been administered. The past history was negative.

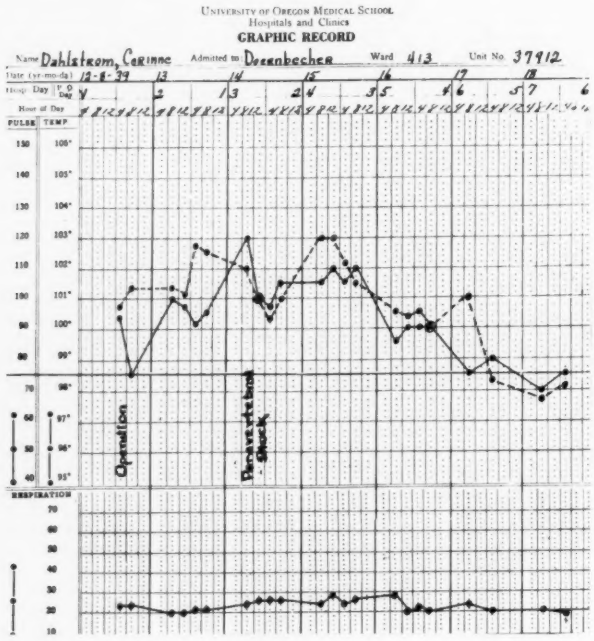


Fig. 3.

Physical Examination: Temperature, 100.4° F.; pulse, 82; respirations, 18. The nose and throat were definitely injected. The chest was resonant and clear throughout. The abdomen was flat and there were marked tenderness and muscle spasm over McBurney's point. Some tenderness was noted high on the right on rectal examination. Urinalysis was negative. White blood count, 12,700; polymorphonuclear leucocytes, 79; small lymphocytes, 16; polymorphonuclear eosinophiles, 1; monocytes, 1; staff cells, 3; sedimentation rate, 15 minutes, 4 mm., and 45 minutes, 28 mm.

Clinical Diagnosis: Acute appendicitis; resolving common cold.

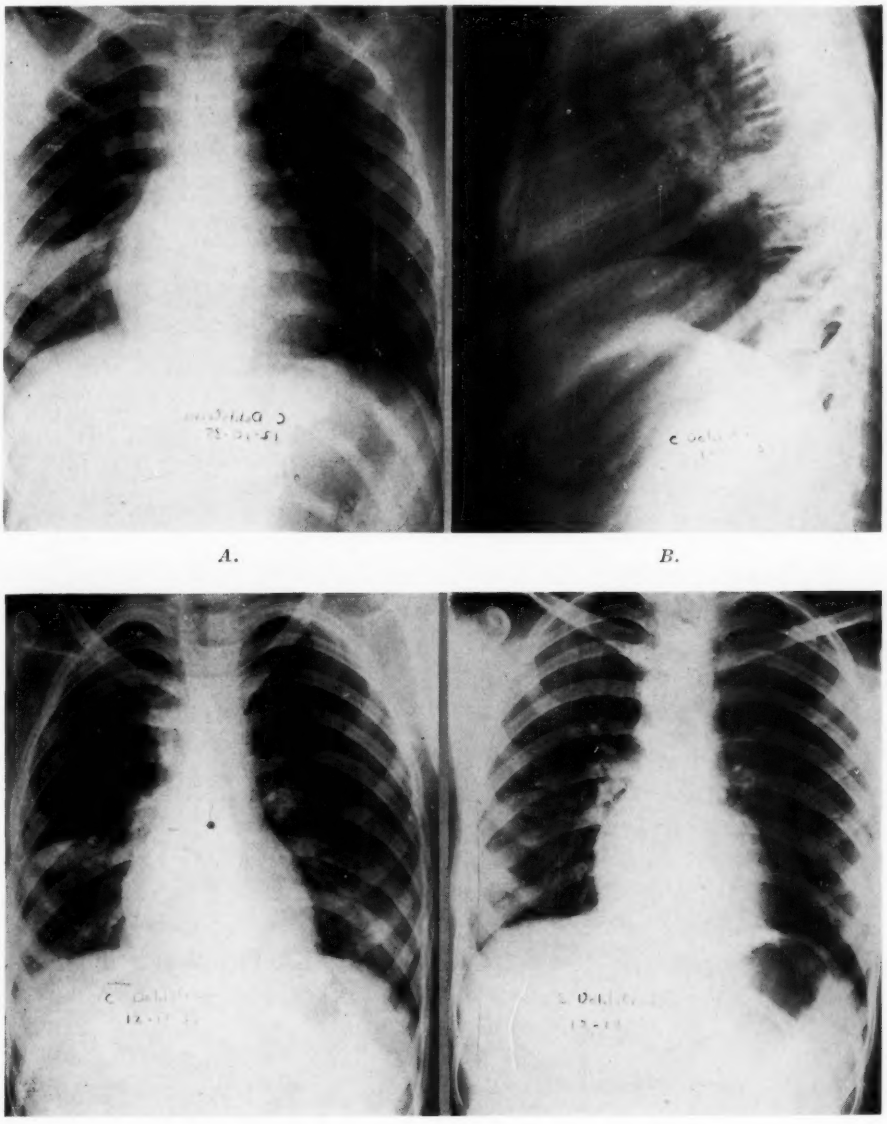
Anesthesia: Cyclopropane, premedication, atropine sulfate gr. $\frac{1}{200}$, sodium phenobarbital gr. $\frac{3}{4}$.

Operation: Appendectomy, McBurney muscle-splitting incision, no drains.

Pathologic Diagnosis: Acute suppurative appendicitis.

Postoperative Course: On the first postoperative day the pulse rose to 118 and

temperature to 101° F. (Fig. 3), but no abnormal symptoms or signs were noted. On the second day the temperature was 103° F.; pulse, 110; and respirations, 26, at 8:00 A.M. At this time a moist productive cough was observed and the patient complained of a great deal of wound pain when coughing. Examination of the chest



A. *B.*

C. *D.*

Fig. 4.—Case 2. *A*, *B*, (Dec. 10, 1939) Posteroanterior and lateral x-rays. There is a triangular shaped area of increased density in the upper portion of the right lower lobe posteriorly. There is no apparent change in the level of the right leaf of the diaphragm, width of the rib interspaces, or the position of the mediastinum. *C*, (Dec. 11, 1939) There is a suggestion of a decrease in the density in the right lower lobe. *D*, (Dec. 14, 1939) The lung fields are clear.

showed decreased expansion on the right, dullness to flatness over the right lower lobe both posteriorly and in the axilla, and distant breath sounds over this area.

An x-ray (Fig. 4*A*) confirmed the diagnosis of right lower lobe atelectasis and a paravertebral procaine block was performed by Dr. John Hutton. Five cubic centimeters of a 1 per cent novocain solution with cobefrin was injected into each of

the following interspaces: dorsal 10, 11, 12, and lumbar 1. Relief of all pain was noted on completion of the block.

Immediately following this procedure the patient began to cough vigorously and expectorated large quantities of mucopurulent sputum. An abdominal binder was applied and the patient was encouraged to cough and was aided in evacuating the secretions by frequent changes in position, percussion over the chest, carbon dioxide inhalations, and ammonium chloride 10 grains every four hours. Three hours later examination revealed some persistent dullness, but the breath tones came through much clearer than previously. During the third and fourth postoperative days the patient continued to cough up moderate amounts of sputum and the chest signs rapidly cleared. On the fifth postoperative day there was no further cough or abnormal signs in the chest.

The possibility of separation of the operative wound following violent coughing must be borne in mind and support of the abdominal wall is indicated. During the period when atelectasis is most likely to occur, the catgut sutures are said to retain their maximum strength. It is true that the added strain of severe coughing occasionally may break or tear out the sutures, but urgent measures are warranted if the more serious pulmonary complications are to be avoided.

I am perfectly aware that the prognosis in postoperative atelectasis is good and resolution occurs in the majority of cases following the usual methods of treatment. The results in these two cases, however, were so striking that there seems to be no doubt that the production of anesthesia in the wound played a significant role in the rapid clearance of symptoms and signs. These cases can hardly be regarded as a conclusive test of a procedure, yet the results observed in the three cases where prophylactic injection was carried out and the two cases reported here convince me that this method of treatment is of practical value.

CONCLUSION

Two cases of postoperative atelectasis which were successfully treated by paravertebral block anesthesia of the operative wound are reported. It is suggested that this procedure which temporarily abolishes pain in the wound and allows for active hyperventilation of the lungs and effective coughing may prove to be an important adjunct to the usual methods now utilized in combating this condition.

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A STUDY OF CAPILLARY PERMEABILITY AND INFLAMMATION IN THE SKIN OF RABBIT GIVEN ADRENALIN*

R. H. RIGDON, M.D., MEMPHIS, TENN.

(From the Department of Pathology, University of Tennessee Pathological Institute†)

PREVIOUS experiments have shown that trypan blue given intravenously will localize and concentrate in areas of skin treated with xylol only when this irritant is applied within a period of less than three hours preceding the injection of the dye. Xylol applied to the normal skin of the rabbit produces hyperemia within twenty to sixty seconds. This hyperemia is subsequently followed by edema and an infiltration of the area with leucocytes.[1] Hyperemia apparently results from a dilatation of the capillaries, venules, and arterioles. Lewis[2] has suggested that such a reaction may result from a local chemical change connected with tissue metabolism and that it is independent of nerve control, either central or local.

Adrenalin injected intradermally produces blanching of the rabbit's skin. Lewis[2] states that this drug acts directly upon the arterioles, capillaries, and venules. Cotton and his associates[3] concur with Lewis. Krogh[4] has expressed the opinion that the effects of adrenalin on the capillaries are produced through its action on the Rouget cells. All physiologists, however, are not in agreement with Krogh. Some hold that the contraction of the capillaries results from the direct action of the drug on the endothelial cells.

Pickrell[5] recently has shown that rabbits narcotized with alcohol or ether show only a few polymorphonuclear leucocytes in areas of skin where pneumococci and aleuronate were injected. Cressman and I,[6] also studying the effects of anesthesia on capillary permeability and inflammation, injected staphylococci, pneumococci, and aleuronate intradermally in rabbits anesthetized with alcohol, ether, and nembutal. Some of our animals narcotized with alcohol or ether did not develop a local inflammatory reaction, while other rabbits given the same anesthesia did show a reaction similar to that in the controls. All the rabbits narcotized with nembutal and injected with the same bacteria, however, developed an inflammatory reaction similar to that in the controls.

It is well known that adrenalin will counteract the swelling induced by the application of irritants to the skin. The edema produced by histamine may also be prevented by the injection of adrenalin. In view of this action of adrenalin on the vascular system it was thought important to study its effect on capillary permeability and the development of an inflammatory reaction at the site of injection of a saline suspension of staphylococci.

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METHODS AND MATERIAL

Rabbits were used in this study. They were carefully shaved twenty-four hours or more before the experiment was begun. Several areas of skin, approximately 2 by 2 cm., were outlined with India ink in those experiments where xylol was used as the local irritant. Duplicate observations were usually made in each animal.

Xylol on a cotton swab was lightly applied to local areas of skin. Care was taken not to traumatize the tissue. A 10 c.c. volume of 0.2 per cent solution of trypan blue was given intravenously immediately following the application of xylol. A record was made of the time when xylol was applied in those experiments where an interval elapsed before the dye was given. The intensity of the local staining of the skin was influenced by the interval between the application of the irritant and the intravenous injection of the dye.

The adrenalin was diluted 1:1000 (Parke-Davis preparation). It was given intravenously in the marginal ear vein in those experiments in which the systemic effects were studied. It was injected intradermally into the blebs produced by the staphylococci when the local effects were studied and also intradermally into the xylol-treated areas of skin when its local effects on capillary permeability were observed. The amount of adrenalin and the frequency with which it was injected varied with different rabbits; usually 0.2 to 0.5 c.c. was given either intradermally or intravenously at each injection. The frequency of the intravenous injections and the amount of adrenalin were influenced by the general appearance of the animal. The quantity of adrenalin given to the different animals is illustrated by subsequent data.

Five-tenths cubic centimeter of an eighteen- to twenty-four-hour broth culture of a nontoxic producing strain of staphylococci was injected intradermally to produce the local areas of inflammation. A quantity of physiologic saline solution equal to the adrenalin was injected into corresponding areas for the control observations on the same animal. The skin at the site of injection of the bacteria was removed after six to eight hours and fixed in a 10 per cent solution of formalin. Sections were stained routinely with hematoxylin and eosin. Select sections were stained by Giemsa's method to demonstrate the bacteria.

CAPILLARY PERMEABILITY IN LOCAL AREAS OF THE RABBIT'S SKIN IN THE PRESENCE OF ADRENALIN INJECTED INTRADERMALLY

The first experiment is a study of the influence of local injections of adrenalin on the localization and concentration of trypan blue in areas of rabbit's skin treated with xylol.

The vascular constriction which normally follows the intradermal injection of adrenalin extends outward for several centimeters from the point of inoculation. This blanched reaction remains for several hours.

The skin, however, becomes reddish brown in color where xylol is applied in the rabbits treated with xylol either before or after the intradermal injection of the adrenalin. This peculiar color appears fifteen to thirty minutes following the application of the xylol. The larger blood vessels in these areas of skin appear dilated. Fig. 1 shows the reddish brown color of the skin in a rabbit given adrenalin intradermally after which xylol was applied to the local areas. Two-tenths cubic centimeter of adrenalin was injected intradermally into the center of Areas 1 and 2. Xylol was then applied to the skin in Area 1 five minutes later. Xylol was also applied at this time to Area 3. The skin was blanched in Areas 1 and 2 at the time the xylol was applied to the

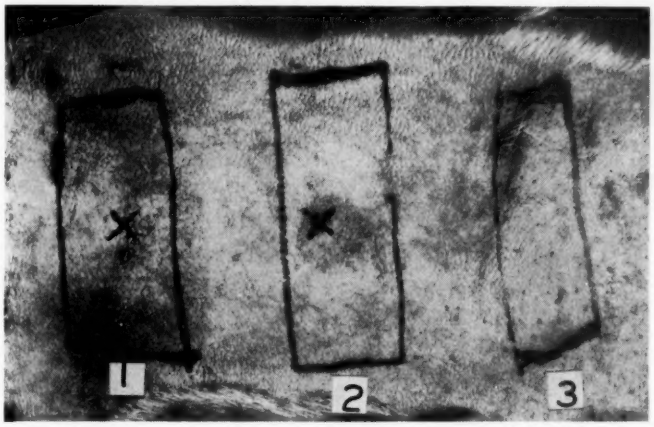

Fig. 1.—Hyperemia in areas of skin treated by the local application of xylol. Two-tenths cubic centimeter adrenalin, 1:1,000 dilution, was injected intradermally at X in Areas 1 and 2. Xylol was applied over Areas 1 and 3 five minutes later. The skin in Area 3 was hyperemic after one minute. Area 3 was hyperemic after fifteen minutes. Area 2 was pale in color at this time. Area 1 was reddish brown in color after two and one-half hours and Areas 2 and 3 were blanched at this time.

skin in Area 3. The skin was also hyperemic in Area 3 five minutes following the application of xylol. This hyperemia decreased, however, after ten to twenty minutes. This diminution in hyperemia apparently followed a diffusion of the adrenalin from Area 2 into Area 3. Such a decrease in the hyperemia as observed here never occurs when the adrenalin is injected in the skin on one side of the rabbit and the xylol is applied to the skin on the opposite side. The skin in Area 1 was slightly reddish brown in color fifteen minutes following the application of the xylol. This color gradually increased in intensity for approximately two hours. Fig. 1 was made two and one-half hours following the application of the xylol.

The following experiment illustrates the changes in color which occur in the skin following the intradermal injection of both adrenalin and saline solution and also the localization and concentration of trypan blue in xylol treated areas of skin in the presence of both saline and adrenalin. Four areas were outlined on the skin on the right (Fig. 2, *1*, *2*, *3*, and *4*) and three on the left (Fig. 2, *5*, *6*, and *7*); 0.3 c.c. of saline

solution was injected intradermally in each of the areas on the right and 0.3 c.c. of adrenalin was injected intradermally into each of the areas on the left. Xylol was applied over Areas 1 and 5 immediately following the injection of the saline solution and the adrenalin. Six hours later xylol was applied to the skin in Areas 2, 4, and 7. Ten cubic centimeters of a 0.2 per cent solution of trypan blue was given intravenously immediately following the last application of xylol. The skin was

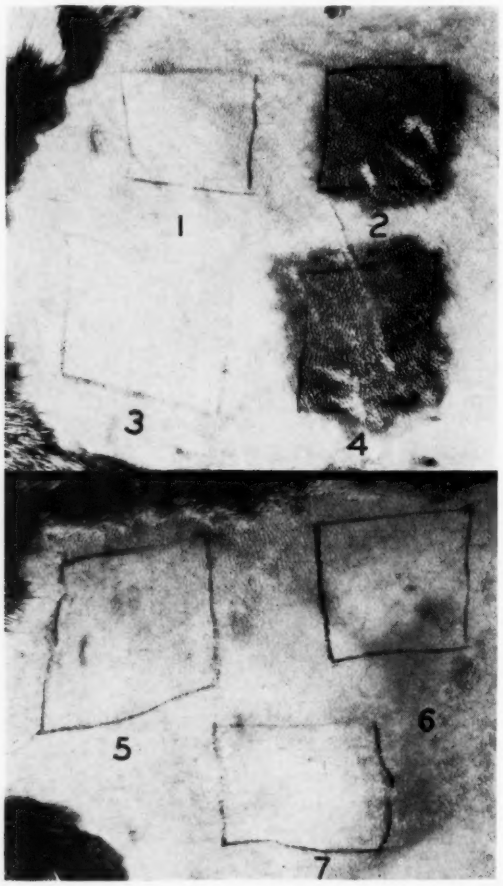

Fig. 2.—Rabbit 481; localization and concentration of trypan blue in areas of rabbit's skin treated with xylol. These areas were previously injected intradermally with 0.3 c.c. saline solution and adrenalin. Areas 1, 2, 3, and 4 were injected with saline solution and Areas 5, 6, and 7 with adrenalin. Xylol was applied over Areas 1 and 5 immediately following the injection of the saline solution and the adrenalin. Xylol was applied over Areas 2, 4, and 7 six hours later. Trypan blue was injected intravenously immediately following the last application of xylol. The dye localized only in Areas 2 and 4. Photographed after thirty minutes.

blanched in Areas 5, 6, and 7 fifteen minutes following the injection of the dye. The skin in Areas 2 and 4 was blue at this time. There was no dye in either Area 1, where saline solution was injected and xylol was applied six hours previously, or in Area 7, where adrenalin was injected and xylol was applied. Trypan blue was present only in Areas 2 and 4 (Fig. 2).

A STUDY OF CAPILLARY PERMEABILITY IN THE SKIN FOLLOWING INTRAVENOUS INJECTIONS OF ADRENALIN

Seven rabbits were used in this second experiment. Xylol was applied to local areas of the skin both before and after the intravenous injection of the adrenalin. The normal skin and the hyperemia produced by xylol blanched following the first intravenous injection of adrenalin. The skin then remained pale throughout the experiment. A protocol on one rabbit illustrates the changes observed in the skin and also the effects produced by adrenalin on the localization and concentration of trypan blue following the local application of xylol.

11:15 A.M.	0.2 c.c. adrenalin intravenously	xylol applied Area 1
11:26	0.2 c.c. adrenalin intravenously	xylol applied Area 2
11:35	0.2 c.c. adrenalin intravenously	xylol applied Area 3
11:40		xylol applied Area 4
11:45	0.2 c.c. adrenalin intravenously	xylol applied Area 5
11:46	15 c.c. of 0.2 per cent trypan blue intravenously	
11:50	Area 5; the skin is light blue	
11:51	Area 4; the skin is light blue	
11:55	Trypan blue is present in Areas 3, 4, and 5	
1:00 P.M.	Trypan blue is present in Areas 1, 2, 3, 4, and 5; there is a progressive decrease in the intensity of color, however, in Area 5 to Area 1	

The dye did not appear in the xylol-treated areas of skin as quickly in some of the rabbits given a larger quantity of adrenalin as it did in the rabbit shown in the above protocol. Such variations, however, are observed in normal rabbits.

All of these experiments show that trypan blue localizes and concentrates in the xylol-treated areas of skin even when there is a quantity of adrenalin in the circulating blood sufficient to produce vascular constriction and inhibits the hyperemia that normally occurs following the local application of xylol.

EFFECTS OF ADRENALIN GIVEN INTRADERMALLY ON THE INFLAMMATORY REACTION PRODUCED BY STAPHYLOCOCCI

The results obtained in the preceding experiments show that adrenalin injected intradermally inhibits the localization and concentration of trypan blue in areas of skin treated with xylol, while adrenalin injected intravenously does not prevent the localization and concentration of trypan blue in areas of skin treated with xylol, although it may inhibit the development of hyperemia and edema.

Seven rabbits were used in the third experiment to study the effect of adrenalin injected intradermally on the local inflammatory reaction produced by staphylococci. Four areas were injected intradermally with the broth culture of staphylococci. Adrenalin was injected immediately in two of these four areas and a similar quantity of saline solution was injected at the same time into the other two areas. The injection of

the adrenalin and the saline solution was repeated after two to four hours.

The skin in the areas injected with staphylococci and saline solution was hyperemic and edematous, while the areas injected with staphylococci and adrenalin usually showed neither hyperemia nor edema after six hours. Histologic studies of the skin in five of the seven rabbits showed a marked diminution in the number of polymorphonuclear leucocytes in the tissues about the staphylococci in the areas injected with adrenalin as compared with the corresponding areas in the same animal injected with saline solution (Fig. 3). Only one rabbit in the group

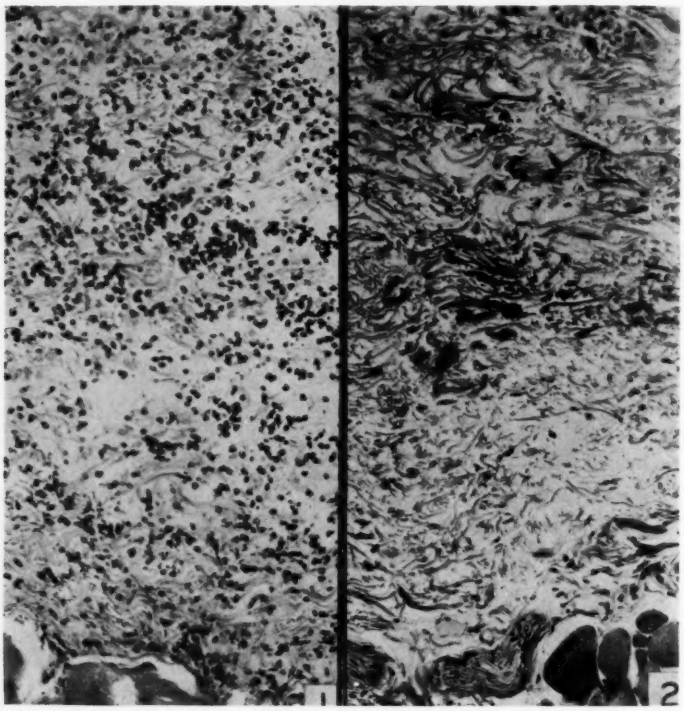

Fig. 3.—Rabbit 510; 0.3 c.c. of an infusion broth culture of staphylococci was given intradermally into four areas. Three-tenths cubic centimeter adrenalin was injected intradermally into two of these blebs immediately thereafter and 0.3 c.c. saline solution was injected at the same time into the other two blebs. The injections of the saline solution and the adrenalin were repeated after three hours. The rabbit was killed eight hours after the staphylococci were injected. Area 1 was injected with saline solution. Area 2 was injected with adrenalin. Note the difference in the number of the leucocytes in the tissue. There are masses of bacteria in Area 2.

showed approximately the same number of leucocytes about the staphylococci in the areas injected with adrenalin as were present in the areas injected with saline solution.

The degree of phagocytosis by individual leucocytes is the same in all sections regardless of the presence of either saline solution or adrenalin.

EFFECT OF INTRAVENOUS INJECTIONS OF ADRENALIN ON THE INFLAMMATORY REACTION PRODUCED BY STAPHYLOCOCCI

In the preceding experiment it was found that the number of leucocytes were decreased in areas of skin where adrenalin was injected intra-

dermally as compared with similar areas of skin injected intradermally with saline solution. Six rabbits were used in this experiment to determine the effect of adrenalin, when given intravenously, on the inflammatory reaction produced by staphylococci. Four rabbits were used in the control group. The frequency of the injections and the amount of adrenalin are illustrated by the following experiment:

9:35 A.M.	0.25 c.c. adrenalin intravenously
9:50	0.2 c.c. adrenalin intravenously
	0.2 c.c. of eighteen-hour infusion broth culture of staphylococci injected intradermally into four areas
10:00	0.2 c.c. adrenalin intravenously
10:20	0.2 c.c. adrenalin intravenously
11:00	0.2 c.c. adrenalin intravenously
11:30	0.2 c.c. adrenalin intravenously
12:00	0.2 c.c. adrenalin intravenously
12:45 P.M.	0.2 c.c. adrenalin intravenously
1:30	0.2 c.c. adrenalin intravenously
2:10	0.2 c.c. adrenalin intravenously
3:10	0.2 c.c. adrenalin intravenously
3:50	Killed, section of the skin injected with staphylococci removed for histologic studies

One of the rabbits in this group died four hours after the experiment was begun. The four areas injected with staphylococci in this animal did not show any hyperemia, edema, or leucocytes. Two of the remaining five rabbits also did not show either hyperemia or edema in the areas of skin injected with the staphylococci. There was some diminution in the number of leucocytes about the organisms in these two animals as compared with the controls. Three of the five rabbits, however, showed both hyperemia, edema, and essentially the same number of leucocytes about the staphylococci as were present about the bacteria in the control animals.

DISCUSSION

These experiments show that trypan blue when given intravenously does not localize in xylol-treated areas of skin when adrenalin is injected intradermally into the same area. These observations suggest that adrenalin either prevents an increase in capillary permeability or this drug produces complete vascular occlusion and thereby prevents any of the dye from reaching the local areas of the skin. The presence of a reddish brown color in the skin thirty minutes following the intradermal injection of adrenalin and the local application of xylol indicates that certain vascular channels must be patent. It is interesting to recall in regard to this reaction that Krogh[4] found the vessels in inflamed tissue unresponsive to adrenalin. The failure of the xylol-treated areas of skin to stain blue following the intravenous injection of trypan blue may be explained, therefore, on a failure of the individual cells to stain rather than upon the failure of the dye to reach the area.

These observations furthermore suggest that the local staining of xylol-treated areas of rabbit's skin by trypan blue may represent not only an increase in the permeability of the capillaries, but also a change occurring in the permeability of the tissue cells. Kline and Winternitz[7] state that the nucleus of a living cell never stains vitally, but as soon as a cell is injured its nucleus absorbs the stain. In view of the observation of Kline and Winternitz it would appear likely that the staining of an area of skin treated with xylol may indicate that some change occurs in the tissue cells following the application of this irritant rather than that the localization of trypan blue indicates only an increase in capillary permeability. Adrenalin apparently may inhibit either the adsorption or the absorption of trypan blue by the cell, although the latter may be injured.

It is interesting to note that in these experiments the hyperemia may be absent in the skin of rabbits in which adrenalin is given intravenously; however, when trypan blue is given intravenously, it localizes and concentrates in these areas of skin recently treated with xylol. This experiment indicates that hyperemia is not essential for the localization and concentration of trypan blue. Previous experiments have also shown that hyperemia and edema are not essential for the localization and concentration of trypan blue.[1] Increase in capillary permeability as shown by the localization of trypan blue may frequently occur, however, in tissues when they are hyperemic and edematous.

The failure of leucocytes to localize in the tissue about staphylococci, when adrenalin is injected intradermally, may be explained by vascular obstruction and a failure of the leucocytes to reach the area. The presence of leucocytes, however, about the staphylococci in the rabbits given adrenalin intravenously may be due to the low concentration of this drug in the local tissue. There was a complete absence of leucocytes about the staphylococci in two rabbits given adrenalin intravenously. It does not appear likely that the absence of leucocytes can be accounted for through the direct effect of adrenalin on the local blood vessels. The amount of adrenalin given to five rabbits in this experiment was sufficient to prevent the occurrence of either hyperemia or edema, but apparently it was an insufficient quantity to prevent the localization of polymorphonuclear leucocytes about the bacteria.

The results obtained in this study would suggest that xylol when applied locally injures tissue cells and such cells may then either adsorb or absorb trypan blue dyes. The period in which the epithelial cells remain permeable to trypan blue is approximately three hours following the application of xylol. The localization and concentration of trypan blue in areas of injury may be an index of increased capillary permeability, but it apparently is also an indication of the duration in which a cell is injured.

SUMMARY

Adrenalin given intradermally or intravenously in large quantities inhibits the development of hyperemia in the skin where xylol is applied. Trypan blue, when given intravenously, fails to localize and to concentrate in areas of rabbit's skin where adrenalin is injected intradermally and xylol is applied locally although the area becomes reddish brown in color after twenty to thirty minutes. Trypan blue localizes and concentrates in areas of the skin treated with xylol when the rabbit is given large quantities of adrenalin intravenously. This variation in the localization of trypan blue in areas of inflammation in rabbits given adrenalin intravenously and intradermally may be explained by a difference in the concentration of the drug in the local areas of tissue.

The number of polymorphonuclear leucocytes is decreased around staphylococci when adrenalin is injected intradermally. There is usually also a diminution in the cellular reaction around groups of staphylococci in those animals given large quantities of adrenalin intravenously. The failure of leucocytes to reach an area of skin infiltrated with staphylococci when the animal is given large quantities of adrenalin intradermally or intravenously apparently may be the result of vascular constriction. The number of leucocytes within the blood vessels in such areas of inflammation is less than in the controls.

The failure of trypan blue to localize in areas of skin where xylol is applied and adrenalin is injected intradermally would suggest that hyperemia and an increase in capillary permeability as manifested by a localization and a concentration of trypan blue do not necessarily have to occur simultaneously. Furthermore, the staining of an area of inflammation in the skin of a rabbit suggests that the permeability of the tissue cells as well as the endothelial cells of the capillaries is changed following the local application of xylol.

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Editorial

Sterile Surgical Supplies

FIFTEEN years ago five patients, operated upon in a single week in a certain New York hospital, died of operative wound infections all of the same general character. From the last case of this series a spore-forming organism of the gas gangrene group was cultivated. It was found to be extremely pathogenic for several species of laboratory animals and produced a powerful specific toxin. The same species of organism was cultivated from two specimens of catgut of the same batch as that used in all of these operations. This seemed to prove beyond peradventure of a doubt that the catgut had not been sterilized and that the catgut was responsible for the infections.

This led to a comprehensive study of the problem of catgut sterility. Raw catgut is contaminated with pathogenic intestinal bacteria which must be killed before it is safe to use as suture material. A survey was made of specimens of catgut purchased on the open market. Twelve and one-half per cent of 174 specimens distributed by seventeen different firms and purchased in eleven different states were found to be contaminated with spore-forming organisms. Seven out of seventeen firms were implicated.

A test for sterility was devised to detect the presence of the most fastidious anaerobe imbedded in any size of catgut preserved in any kind of suspending fluid if it has not been destroyed in the sterilizing process. It is not feasible for surgeons and it should not be necessary for hospitals to test the sterility of the catgut which they use. The responsibility lies with the manufacturers of the product. It is obvious that, if every catgut firm subjected specimens from every sterilizing batch of catgut to such a sterility test before releasing the catgut for sale, it would hardly be possible for hospitals or doctors to purchase and use any unsterile catgut.

How could catgut firms be made to test their product and store it long enough to be sure of its sterility before offering it for sale? The only practical proposal which was made to accomplish this feat was to set up a reliable Control. Specimens of catgut would have to be purchased on the open market and be tested in some laboratory according to the sterility test devised. The Control should then immediately stop the further distribution of any faulty material. This would mean confiscating all of the unsterile batch and making it known to the surgical profession that the product was unreliable.

The Bureau of Standards was asked to undertake such a control but refused to do so. In like manner the National Institute of Health was

unwilling to assume that responsibility. During the course of the next ten years, repeated efforts were made to get some recognized authority to establish a control over sterile surgical products. The Hospital Standardization Committee of the American College of Surgeons was appealed to, but claimed that they had no funds or laboratory space which could be used for the purpose. The American Medical Association was therefore called upon for help and appointed a Committee from members of the Surgical Section of the Association to study the problem. This Committee believed that it would be wise to set up not only a standard of sterility for catgut but standards of other desirable physical properties, such as size, tensile strength, and absorbability. The Committee studied the problem carefully, recommended a complete set of catgut standards, and advised the American Medical Association to set up a continuous control. This proposal was turned down by the A. M. A. The reason given was that it was thought that the situation had improved to such an extent that there was no longer any need for such a control and therefore the expense would be unwarranted. The matter was referred to the Council on Pharmacy and Chemistry. This body assigned one of its members to investigate the situation and after a year's study it was found that conditions had considerably improved in the course of ten years, but that unsterile catgut was still being sold.

The New York Academy of Medicine's Committee on Public Health Relations then became interested and promised to establish some kind of control if it were not possible to persuade some governmental agency to do so. About this time the Food and Drugs Administration was asked to examine a lot of suspected catgut and found it to be unsterile. They at once confiscated the goods and began a court action against the offending firm, on the assumption that the Pure Food and Drugs Act had been violated. Their contention was upheld by the court, but the case was appealed on the basis of the assertion that catgut was not a drug and therefore the sale of unsterile catgut was not a violation of the Act.

This uncertainty was clarified when the new Food and Drugs Act was formulated and adopted last year. It is specifically stated therein that not only catgut but also all sterile surgical supplies, including cotton, gauze compresses, bandages, bandaids, first aid dressings, adhesive plaster, plaster of Paris, etc., are drugs because they are used in the treatment of disease. If they are put out as sterile goods to be introduced into the human body or applied to its surface, there must be no contamination or adulteration with bacteria or any other extraneous matter.

The U. S. Pharmacopoeia has, for some time, been accepted as the standard authority for pure drugs. With the passage of the law it became the duty of the Revision Committee of the Pharmacopoeia to set up standards for these sterile surgical supplies. An advisory board

was selected to help in establishing these standards. This Board is composed of Dr. George McCoy, Dr. Harrison, of the National Institute of Health, and Dr. Louis Gershenfeld, of Philadelphia, with Dr. Arthur Shipley and myself representing the surgical profession. The Board has met on numerous occasions with representatives of the Bureau of Standards, the Food and Drugs Administration, the Army and Navy supply officers, and other government experts as well as with representatives of the manufacturers. Last year standards were set up for catgut and for absorbent cotton. Also a test for the sterility of all solid goods was established based on the sterility test which had been developed for catgut. These standards were incorporated into the supplement of the eleventh decennial revision of the Pharmacopoeia, which was published on Jan. 1, 1940, and will go into effect on Jan. 1, 1940. On that date the Food and Drugs Administration began its control over sterile surgical supplies. The manufacturers of these products will soon have to check the sterility of all of their sterile goods and strictly conform to the standards or risk the confiscation of these goods and court action. It is hoped that these measures will effectively banish unsterile catgut and other harmful surgical supplies from the market and that the surgical profession may purchase and use these goods with absolute confidence that they are working with sterile material and not running the risk of infection by their use. Thus will the labors of fifteen years be amply rewarded.

While the establishment of this control is the culmination of fifteen years of effort, we do not feel that this is to be the end of our labors. The standards which have been set up are not complete. For example the qualities of catgut next most important to sterility, which is an absolute requirement, are tensile strength and absorbability. Standards have been set up for tensile strength, but we expect the manufacturers to improve this quality and compete with one another in this effort. If we can get stronger catgut, we may employ finer sizes to do the same work we are now asking large sizes to do. When this is done we will undoubtedly want to raise the standard of tensile strength. With regard to absorbability the material available shows the greatest possible diversity. Some catgut firms have been putting out what they call ten-, twenty-, or forty-day chromic catgut, but when we asked them just what these figures meant some claimed that these sutures would hold tissues together for that length of time while others said that they meant the suture would resist complete digestion for that length of time, which is quite a different matter. Other firms contented themselves with the terms soft, medium, and hard to indicate the degree of tanning or chromicizing. It has been impossible to devise animal tests for catgut absorbability which duplicate conditions in the human body because so many factors are involved; for example, the tension of the sutures, the various tissues in which they are placed, and

the forces to which they are subjected during respiration, vomiting, hiccoughing, abdominal distention, and so forth. For that reason a test tube digestion test under controlled conditions has been devised which is being given a thorough try out this year with the Surgeon General's consent in the U. S. Army testing laboratories. We hope that it will at least serve as a basis for judging the uniformity of catgut from the point of digestibility in the test tube and that it may later be correlated with absorbability in the human body. Not until this and standards for the other sterile surgical supplies are satisfactory will they be incorporated into the Pharmacopoeia. It is hoped that this will have the enthusiastic support of all surgeons and all hospitals and that they will purchase only those products which meet the required standards.

—*Frank L. Meleney, M.D.*
New York, N. Y.

Recent Advances in Surgery

CONDUCTED BY ALFRED BLALOCK, M.D.

ACUTE PERFORATED GASTRODUODENAL ULCERATION

A STATISTICAL ANALYSIS AND REVIEW OF THE LITERATURE

MICHAEL DEBAKEY, M.D., NEW ORLEANS, LA.

(From the Department of Surgery, School of Medicine, Tulane University)

ACUTE perforation is undoubtedly one of the most serious and important complications of gastroduodenal ulceration. Its frequent and ubiquitous occurrence, its bewildering pathogenesis, and its difficult and controversial management have made it a subject of perennial inquiry. The increasing voluminous literature that has accumulated on this subject clearly reflects its intriguing interest. Moreover, in spite of a better comprehension of peptic ulceration, it would appear that its incidence is increasing. Of even greater significance is the fact that, notwithstanding incessant experimental and clinical investigations and distinct improvements in surgical management, the relatively high mortality during the past decade has decreased insignificantly. Thus, in 1930 the general mortality incidence of the collected reports was 26.1 per cent and in 1939 it was 23.7 per cent, a difference of only 2.4 per cent or a decrease of approximately 9 per cent. With these facts in mind, a review of the literature and a critical analysis of the subject is considered opportune. An attempt has been made to limit the review to acute perforations and not to consider the subacute or the "forme fruste" types[374, 436, 572, 600, 606, 607] nor perforation of malignant lesions.[4, 108, 215, 236, 237, 259, 371, 406] Statistical analysis of the collected cases has also been limited to those reported during the past decade. Accordingly, over 20,000 cases have been collected from the literature representing reports from various parts of the world.

Historical Considerations.—One of the earliest accounts as well as one of the most graphic clinical descriptions of perforated gastric ulcer is that written by Madame de la Fayette of the fatal illness of her royal mistress, Princess Henrietta Ann, Duchess of Orleans, daughter of King Charles I of England and wife of Philip, brother of Louis XIV, King of France.[385] The frail Princess had suffered with abdominal discomfort for some time previously but on the day of her death, in 1670, immediately after drinking chicory water, she complained of excruciating abdominal pain. Rapidly sinking into a collapsed state she died nine hours later. The results of the autopsy have been preserved in the memoirs of the English king's surgeon who was

present at the post mortem. Fetid gas was present in the peritoneal cavity as was a "sanious putrid, yellow, watery fluid mixed with oil." The intestine was greatly distended and discolored and the stomach showed no lesion except a small hole in the middle and anterior portion "which had been accidently produced by the surgeon with the point of his scissors."[306, 367]

According to Leube,[361] Grassius[250] in 1695 described a case of perforated gastric ulcer and Lenepneu[359] quotes de Muralto[151] in 1688 as observing duodenal perforations in a soldier.

One of the earliest authentic cases of perforated gastric ulcer is the admirable presentation of Christopher Rawlinson[537] before the Royal Society in 1729. The patient, who had complained of severe abdominal pain for several years, died in the hospital "without the least amendment to his disease." At autopsy, upon opening the abdomen, "there flowed out a whitish Liquor . . ." and "When we came to the Stomach we found it perforated in its upper Part, about the middle Space betwixt the Two Orifices, wide enough to contain the End of one's finger."

Perhaps the earliest description of a case of perforated duodenal ulcer is that reported by Hamberger[270] in 1747. The patient, a married maid servant, complaining of severe abdominal pain, died implicating her husband in foul play. A judicial autopsy was, therefore, performed and a perforated ulcer observed in the duodenum "six lines away from the pylorus."

Jacopo Penada, of Padua,[501] in 1793 published a volume of 146 pages, 23 of which were devoted to the accurate description of a fatal case of perforated duodenal ulcer in a butcher 35 years of age. This patient, who was "gifted with tremendous strength" and addicted to "continuous debauchery," suffered from epigastric pain following meals for twenty-six days before his death. He was suddenly seized with excruciating pain which rapidly spread over the entire abdomen three days prior to his death. At autopsy there was found a generalized peritonitis, an empty stomach, and a perforated ulcer in the duodenum "four finger breadths below the pylorus."

Cases of perforated ulcer were reported by Neumann[455] in 1802 and by Gerard[235] in 1804. In 1817 Travers[649] and Crampton[127] published several cases of perforated ulcers which they had observed, and Travers[649] gave an excellent description of the diagnostic symptoms and signs. Following this a number of other reports[1, 23, 296, 446, 549] appeared and in 1856 Brinton's[79] meritorious and comprehensive presentation was published. In addition to bringing the literature up to date, this author analyzed statistical data on the subject of gastric ulcer and its complications which are surprisingly similar to present-day statistics. Other extensive reviews in monographic form were published by Müller[445] in 1860 and by Krauss[341] in 1865. During the

latter half of the nineteenth century, numerous isolated cases appeared in the literature[30, 66, 107, 111, 112, 196, 335, 370, 673] as well as a number of theses.[7, 576, 650] In 1875 O'Hara[475] presented before the Philadelphia Pathologic Society a case of perforated duodenal ulcer, which is believed to be the first recorded case in America.

The first operation for perforated ulcer was probably performed by Mikulicz[419] in 1880. He repaired the opening by simple suture, but the patient died three hours later in shock. Roux[554] in 1883 and Mackenzie[378] in 1888 also operated upon cases which terminated fatally. In 1892 Kreige[344] reported the first successful suture, performed by Heussner, of a perforated gastric ulcer.

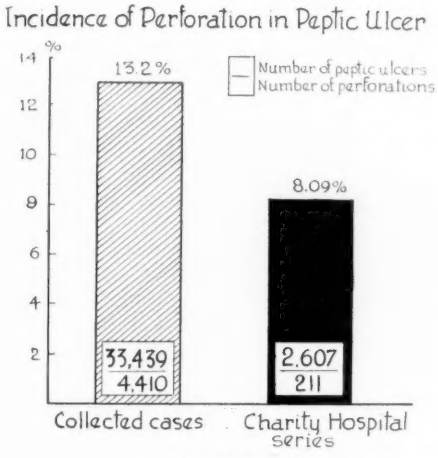
Two years later Barling[31] and McClaren[380] each reported cases operated upon successfully. Dean[139] in 1894, Herczel[283] in 1895, and Dunn[170] in 1896 recorded successful operations for perforated duodenal ulcer. In 1894 Gould[246] collected six cases successfully operated upon. Weir,[683] in 1896, is believed to be the first American surgeon to operate upon a case of perforated gastric ulcer and Atherton,[16] in 1894, the first to operate upon one successfully. In 1892 Hall[268] collected six cases in which spontaneous recovery occurred. Braun[68] in 1897 introduced the performance of gastroenterostomy in addition to the suture of perforated ulcer and Dowden[160] in 1909 reported the procedure of excising the ulcer along the longitudinal axis and suturing the opening thus formed along the transverse axis. In 1919 von Haberer[265] advised resection in perforated ulcer.

Incidence.—Perforation has been estimated to occur in incidences varying from 1.2 per cent[299] to 28.1 per cent[205, 449] of all gastroduodenal ulceration. Speck,[620] who reviewed the literature up to 1923, recorded an incidence of 10 per cent and Deaver[141] stated that it occurs in about 20 per cent. Bager[21] found that perforation occurred in 18.1 per cent of 9,474 cases of gastroduodenal ulcers collected from various Swedish hospitals. At the Charity Hospital in New Orleans, during the ten-year period 1929 to 1938 inclusive, there were admitted 2,607 cases diagnosed as peptic ulcer and of this number there were 211 (8.09 per cent) cases of acute perforated ulcer.[474] In a series of 33,439 cases of gastroduodenal ulcers collected from the literature,[713] there were 4,410 (13.2 per cent) cases of perforation (Graph I).

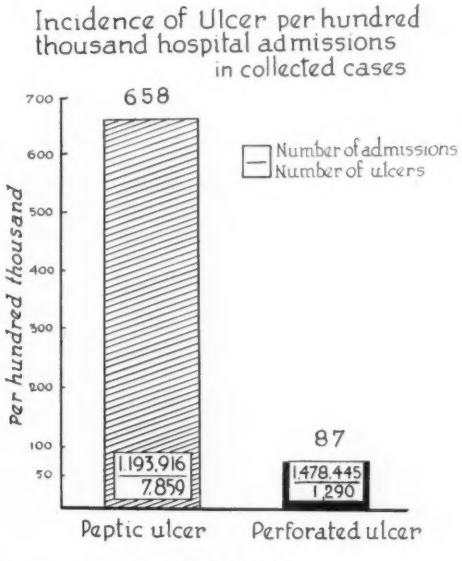
The incidence of acute perforated ulcer in hospital admissions also varies considerably in the reported statistics. Thus, Segal and Scott[585] found the low incidence of 0.019 per cent in 110,450 admissions and Garver[229, 230] reported the high incidence of 0.982 per cent in 4,377 admissions. During the ten-year period 1929 to 1938 inclusive at the Charity Hospital in New Orleans, there were 544,801 hospital admissions and of this number there were 2,607 (0.478 per cent) cases of peptic ulcer and 211 (0.038 per cent) cases of acute perforated ulcers.[474] In a collected series of 1,193,916 hospital admissions[55, 105, 229, 230, 474, 585, 608, 640] there

were found 7,859 cases of gastroduodenal ulceration, an incidence of 658 per 100,000 (Graph II). In a collected series of 1,478,445 hospital admissions[55, 105, 229, 230, 264, 303, 330, 474, 526, 585, 608, 640] there were 1,290 cases of perforations, an incidence of 87 per 100,000 admissions (Graph II).

Of particular significance in this regard is the fact that, whereas peptic ulcer in general seems to be increasing, the frequency of acute perforation is becoming disproportionately greater. During the past decade numerous authors[97, 104, 173, 292, 355, 708, 711] in various parts of the world have directed attention to this alarming fact. Semb[588] reported that at the Oslo Municipal Hospital there was a definite annual increase from 2 cases in 1912 to 20 cases in 1928. Similarly Chang[105] in China stated that there was a consistent increase from 1 case per year



Graph I.

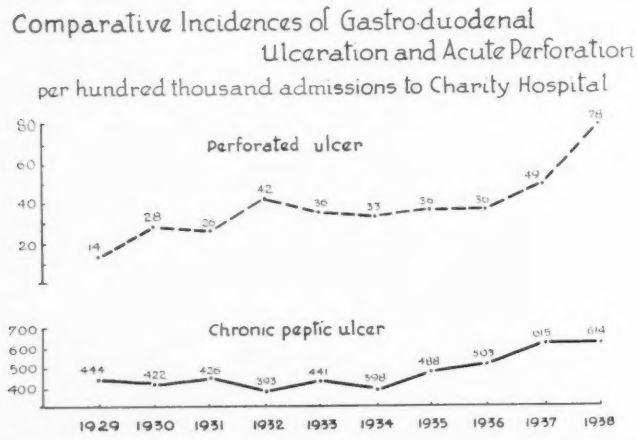


Graph II.

Graph I.—Incidence of perforation in gastroduodenal ulceration.
Graph II.—Respective incidences of gastroduodenal ulceration and perforation per 100,000 hospital admissions.

in 1912 to 7 in 1936. Richardson[545] found that during the ten-year period 1896 to 1905 there were only 18 cases, but during a similar period, 1906 to 1915, there were 64 cases. Wilmoth[696] in France found that during the nine-year period 1920 to 1928 there were 18 cases; whereas, during the one and one-half-year period 1929 through the first half of 1930, there were 16 cases. Similarly Mollo[424] found only 6 cases between 1922 and 1928 and 32 cases between 1928 and 1932. Schilling[568] reported only 71 cases during the ten-year period 1912 to 1922 and 194 cases during the two-year period 1923 to 1924. Dansey[136] stated that there were 62 cases from 1915 to 1919 and 97 cases from 1920 to 1925. Nicolas[458] recorded that, although there were 11 cases in 1927, during the preceding eleven years there was a total of only 39

cases. Stipa[627] observed that prior to 1928 there was an average of 1 case per year; whereas, in 1929 there were 3 cases and in 1936, 29 cases. Barbera[29] found that during the three successive five-year periods from 1915 to 1929, there were 16, 27, and 57 cases respectively. Troutt[655] concluded from a statistical analysis that peptic ulcer is steadily on the increase in the United States Army. He found that the incidence of perforation in gastroduodenal ulceration increased from a ratio of 1 to 41 ulcers in 1922 to 1 to 7 in 1926, an increase of almost sevenfold in a period of five years. Shawan[593] analyzed the incidence at the Receiving Hospital of Detroit over an eighteen-year period, 1920 to 1937 inclusive, and found an almost consistent annual increase of from 2 cases in 1920 to 33 cases in 1937. Similarly, Judine[323] in Russia studied the incidence during a thirteen-year period, 1925 to 1937 inclusive, and found an annual increase from 25 cases in 1925 to 273 in 1933 with a gradual decline to 83 in 1937. Bager[21] found in an



Graph III.—Comparative incidences of gastroduodenal ulceration and acute perforation per 100,000 admissions at the Charity Hospital in New Orleans during the ten-year period, 1929 to 1938 inclusive. It is obvious that there is a proportionately greater increase in the incidence of perforation than in ulceration in general.

analysis of 1,767 cases admitted to 50 hospitals in Sweden that during the five-year period 1911 to 1915 inclusive there were 332 cases, and that during the two successive five-year periods the number of cases increased to 470 and 693 respectively. At the Charity Hospital in New Orleans the incidence of gastroduodenal ulceration increased gradually from 443.7 per 100,000 admissions in 1929 to 614.3 in 1938. However, the comparable figures for acute perforated ulcer were 13.9 and 78.3. Thus, during this ten-year period the increase in gastroduodenal ulceration was less than one and one-half times; whereas, the increase in acute perforated ulcer was almost six times (Graph III).

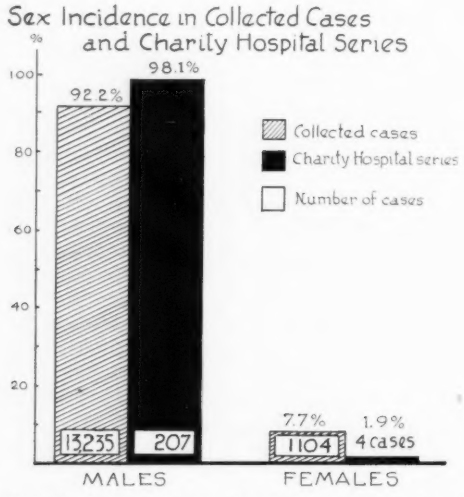
The cause for this increased frequency has not been satisfactorily explained. Henle[280] is of the opinion that alcohol and tobacco play prominent roles. The increased use of alcohol since the repeal of the

Eighteenth Amendment has also been suggested as a factor in the United States. The consequences of the general economic depression since 1929 have been suggested as an explanation in a rather conflicting manner. Hull[303] believes that the depression and the consequent individual neglect of diet and oral and general hygiene may be a factor in the increased incidence. On the other hand, Sallick[560] who observed a decreased incidence from 1929 to 1932, expressed the opinion that the "increased unemployment since 1929 with less hurry and physical strain on those engaged at their occupation may be factors of some importance in the lessened incidence." In Germany the increased incidence observed by Zuckschwerdt and Eck[711] has been explained by these authors as possibly due to improved nutritional conditions. Troutt,[655] who observed a distinct increase in perforated ulcers among soldiers in Hawaii, believes that the responsible factors are "fundamentally psychic and include nostalgia, mental depression, lowered nerve tone, excessive smoking, and, most important of all, the excessive use of impure alcoholic beverages. Obviously, explanations for the increased incidence can be only speculative. I am in complete accord with Ochsner[469] that the increased use of tobacco plays a significant role. Of particular interest in this connection are the intriguing observations of Bager,[21] who obtained corresponding increases in the annual tobacco consumption incidences and the annual perforated ulcer incidence in Sweden. Bager believes tobacco smoking is an important influential factor.

Sex Incidence.—Acute perforated ulcer occurs predominantly in the male sex. Of particular interest in this regard, however, is the fact that apparently this predominance in the male sex is becoming more marked. In 1856 Brinton[79] found that in a series of 234 cases the incidence of the female sex was 68.4 per cent. In 1903 Brunner[84] reported a 66.4 per cent incidence in the female sex in a series of 423 cases. Petrén[508] in 1911 reported a 48 per cent incidence in females. Morley[429] in 1913 reported 112 cases, of which 32.1 per cent were females. In 1914 Shoemaker[597] reported an incidence of 52 per cent in women. Norrein[464] in 1915 found a 40 per cent incidence in the female sex and Hertz[285] in 1918, 39.5 per cent. In a large collected series Moynihan[438] in 1910 found the incidence in women to be 26.9 per cent and Schwarz[577] in 1925, 24.7 per cent. Speck[620] analyzed the cases admitted between 1900 and 1922 and found that from 1900 to 1910 the incidence in the female sex was 73.6 per cent; between 1911 and 1920, 24 per cent; and between 1921 and 1922, none. Bager[21] showed graphically that during the period 1911 to 1925, whereas there was little or no increase in the incidence of perforation in the female sex, there was a steady and rapid rise in the male sex. Thus, it can be observed that early statistics showed an almost equal and, in some instances, even greater incidence in females. Jennings[310] has recently

discussed this peculiar fact and states that it is difficult to determine exactly when perforations in young women ceased to be so frequent. He found that previous to 1900 the incidence in females was greater in the early age group and that subsequently there was a shift to an older age group. He believes that another explanation for the greater incidence of perforation in females previously is that it "must have been due to something in the mode of life" at that period.

More recent statistics show a definite preponderance in the male. During the decade 1920 to 1929, a number of authors[714] reported incidences varying from 75.7 per cent to 100 per cent in males. During the past decade, larger series have been reported, with incidences usually over 90 per cent in the male. In 1929 Dineen[154] reported 142 cases with 97.2 per cent in males; in 1931 Zuckschwerdt and Eck,[711] 117 cases with 93.1 per cent males; in 1933 Graves,[253] 141 cases with

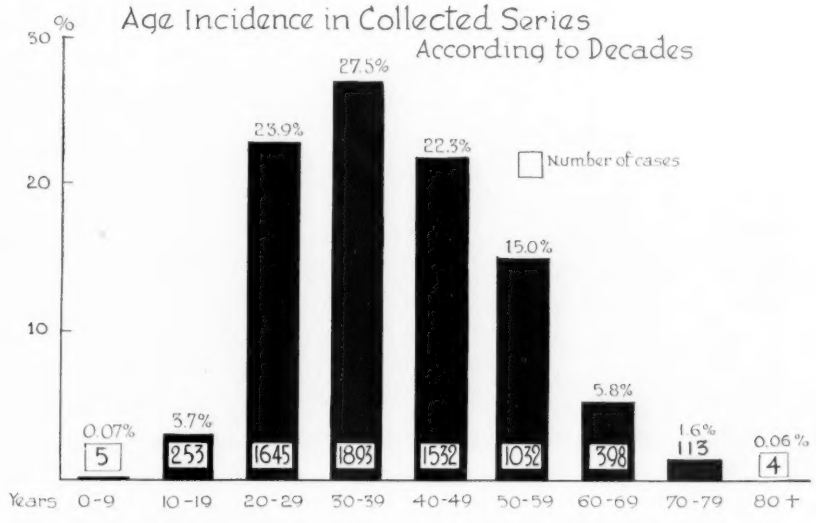


Graph IV.—Sex incidence in perforated gastroduodenal ulceration.

97.9 per cent males, Schilling[568] in 1935, 265 cases with 84.9 per cent males; Thompson[640] in 1937, 500 cases with 94.2 per cent males; Herzberg[286] in 1938, 410 cases with 93.7 per cent males; Judine[323] in 1939, 1,355 cases with 98.1 per cent males. In the Charity Hospital series there were 207 (98.1 per cent) males and 4 (1.9 per cent) females.[474] In a collected series of 14,339 cases[715] there were 13,235 (92.2 per cent) males and 1,104 (7.7 per cent) females (Graph IV). These statistics reveal clearly that acute perforated ulcer in more recent years occurs with overwhelming predominance in the male sex. No satisfactory explanation for this fact exists. Peptic ulcer in general occurs more frequently in the male. Attention has been directed also to the fact that the predominance of males is less marked in gastric ulcer than in duodenal ulcer. Obviously, these statistics suggest a relationship of sex hormones to peptic ulcer and this question has been investigated

by Sandweiss and co-workers,[562] who observed that in a collected series of 70,310 consecutive admissions of pregnant women during a ten-year period there was only 1 true case of active peptic ulcer. They discussed the possibility of a protective mechanism provided by the monthly rhythmic interplay between pituitary and ovary. They[193] made preliminary observations on Mann-Williamson dogs which indicated that urine from normal individuals contains a "protective factor" against this type of ulcer and this "protective factor" is definitely absent in urine of patients having peptic ulcer.

Age Incidence.—The age incidence in acute perforated ulcer is approximately the same as peptic ulcer in general; i.e., it occurs with greatest frequency during adult life in the third, fourth, and fifth decades. Various investigators have reported incidences varying from 64.6 per cent to 87.1 per cent[21, 323, 535, 583, 588, 592, 629, 640] in these decades. In a collected series of 6,875 cases 73.1 per cent occurred in the third



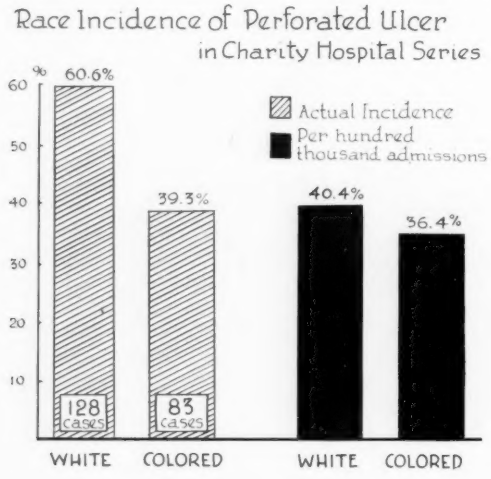
Graph V.—Age incidence in perforated gastroduodenal ulceration.

to the fifth decades inclusive, only 4 per cent in the first and second decades, and approximately 22 per cent in patients over 50 years of age[716] (Graph V). In the Charity Hospital series these respective incidences were 78.2, 3.7, and 18.3 per cent.[474]

Acute perforated ulcer in infants and children occurs very rarely. However, cases have been recorded in infants 22 months of age,[598] 18 months,[698] and even under 1 year of age.[2, 80, 208, 256, 274, 279, 362, 570, 621, 706] Somerford[615] reported a case in an infant 14 days of age; Thelander and Mathes,[638] in one 10 days of age; Seinsheimer[586] and Butka[92] each, in infants 4 days old; and Stern and co-workers,[625] in one 2 days old. It is not generally realized that peptic ulcer does occur in children and infants. In 1913 Schmidt[570] found that in 1,019

post-mortem examinations in infants less than 1 year of age there were 20 (2 per cent). This subject has recently been discussed by Kennedy.[332] Unusual cases have also been reported at the other age extreme. Dunbar[168] had one case in a patient 74 years of age; Montgomery,[427] in one 75 years of age; Hinton,[291] in one 80 years of age; Sandell,[561] in one 82 years old; and Kelly,[330] in one 84 years of age. Thompson[640] reported 3 cases in a series of 500 patients over 81 years of age.

Race Incidence.—The racial factor has been considered as playing a role in gastroduodenal ulceration. It is the common belief that acute perforated ulcer occurs relatively infrequently in the negro.[67, 216, 631] Dineen[154] in a series of 142 cases found only 3 negroes. Others have reported incidences of 2.3 per cent,[536] 3.8 per cent,[89] 6.9 per cent,[697] 7.1 per cent,[404, 613] 10 per cent,[82] 16.2 per cent,[179] 20 per cent,[78, 303, 656] 24.2 per cent,[157] and 25 per cent.[493] Thompson[640] found that of 352 cases 332 (94.3 per cent) were in the white, 17 (4.8 per cent) in the



Graph VI.—Comparative race incidences of perforated gastroduodenal ulceration as based upon actual number of perforations and corresponding per 100,000 admissions.

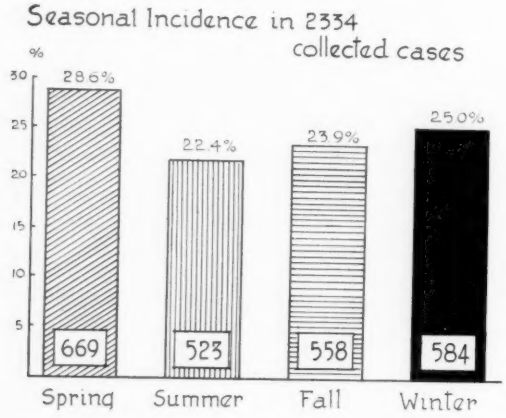
negro, 1 (0.28 per cent) in the Indian, and 2 (0.57 per cent) in the Mongolian races. Such incidences, however, are not faithful representations as the white race in general in these various clinics is preponderant. Therefore, a more accurate estimate could be obtained by incidences based upon hospital admissions. Thus, Thompson[640] found that the incidence of perforations per 100,000 admissions was 157 in the Caucasian, 156 in the Mongolian, 121 in the negro, and 746 in the Indian races. According to Garver,[229] the relative incidences of perforated ulcer in the white and negro races per 100,000 admissions at the Grady Hospital in Atlanta were 745 and 982 respectively. Similarly, Sharon[590] found these respective figures for peptic ulcer to

be 423 and 347. In the Charity Hospital series of 211 cases there were 128 (60.6 per cent) white and 83 (39.3 per cent) colored patients.[474] Whereas, this would seem to indicate a greater incidence in the white race, it must be realized that the incidence of total admissions of white patients is greater than that of colored. Accordingly, if the respective incidences are based on corresponding per 100,000 admissions, these figures are found to be 40.4 and 36.4 (Graph VI). Thus it may be observed that acute perforated ulcer occurs almost as frequently in the negro as in the white race.

This problem has recently been reviewed by Sharon,[590] who found that the incidence of duodenal ulcer in the negro was slightly lower than in the whites. It is considered inopportune to attempt here a detailed discussion of the anthropological, anatomical, psychological, and environmental factors involved in this question. The reader interested in this phase of the subject may refer to the extensive exposition of Matas[394] on the surgical peculiarities of the negro and the recent review of Sharon.[590]

In a recent report Chang[105] has shown that the incidence of this condition among the Chinese is approximately the same as in this country. Chang and Chang[106] emphasize the fact that the assertion by Finnigan[207] that peptic ulcer occurs less frequently in the Oriental races is entirely without substantial foundation. They found that in the Peiping Union Medical College Hospital during the period 1921 to 1926 inclusive the incidence of peptic ulcer was 0.86 per cent of the medical and surgical admissions to the hospital. They also found that the incidence of perforation was 10.4 per cent. This question has recently been reviewed by Necheles.[451] McCarrison[398] directed attention to the fact that peptic ulcer is practically unknown in northern India but occurs with great frequency in southern India. It is his opinion that nutritional or dietitic factors were probably responsible and that in the south there was a lack of essential vitamins. Bergsma[43] studied this problem among negroes and the black people of Abyssinia and stated that the relative frequency of ulcer among the Abyssinians was exceptional to the usual infrequency among negroes and he believed this to be attributable to the high red pepper content of the diet of these people. In 1921 Snijders and Straub[612] directed attention to the fact that a striking difference in the frequency of gastric ulcer exists between the Malays and Chinese in Java and Sumatra. Bonne and associates[60] studied the peculiar phenomenon that a higher incidence of major gastric lesions occurred in the Chinese than in the Malays and were unable to demonstrate any responsible factor. It may be concluded from this brief discussion that, whereas a racial factor may exist in acute perforated ulcer, it is difficult to define it and any discussion can be only conjectural.

Seasonal Incidence.—That there may exist a definite seasonal influence in gastroduodenal ulceration has never been definitely established. A number of investigators have directed attention to the possibility of a seasonal factor in acute perforated ulcer. Whereas many of these[303, 324, 542, 616] have expressed the opinion that acute perforated ulcer occurs most frequently in the spring and fall and others[59, 286] have considered autumn and winter or winter[355] and summer,[163] some[154, 277, 560] have expressed the belief that there are no significant seasonal incidences. In a collected series of 2,334 cases in which the seasonal incidence was stated, 28.6 per cent occurred in the spring, 22.4 per cent in the summer, 23.9 per cent in the fall, and 25 per cent in the winter (Graph VII).[717] In the Charity Hospital series of cases these respective figures were 24.6, 24.2, 27.9, and 23.2 per cent.[474] Whereas in the collected series the incidence was slightly greater in the spring, in the latter group it was slightly greater in the fall. However, the variations in both series were very slight and probably statistically insignificant. Judine,[323] who observed a slightly increased frequency in the spring and fall, expressed the opinion that a possible explanation lies in the grippe epidemics during these seasons and the inability to obtain adequate amounts of fresh fruits and vegetables during the winter months with consequent diminution in vitamins, which would be likely to become manifest towards the end of winter or the beginning of spring.



Graph VII.—Seasonal incidences in perforated gastroduodenal ulceration.

Occupation.—The significance of occupation in perforated ulcer is not clear. The common belief that the high tensioned, energetic executive is the likely candidate for perforated ulcer is not corroborated by statistical analysis. Indeed, it would appear from the reported statistics that the laboring classes, both skilled and unskilled, are by far the commonest group. Various investigators have reported incidences in this class of 50 per cent,[355] 63 per cent,[711] 69 per cent,[677]

and 75 per cent.[192] Thompson[640] in an analysis of 453 cases found that only 19.8 per cent were mental workers or of the executive class; whereas, 58.7 per cent were skilled laborers and 15.4 per cent were unskilled laborers. Read[540] reports 48.4 per cent of his series were skilled laborers and 29.7 per cent were unskilled. Tilton[643] records 34 per cent laborers and 66 per cent in the sedentary class and Chang's[105] figures were somewhat similar, being 37.8 per cent and 62.2 per cent respectively. In the Charity Hospital series of 211 cases 72.9 per cent were laborers, 21.3 per cent were sedentary workers, 1.4 per cent were household workers, and 4.3 per cent were unemployed. Shelley[594] made the interesting observation that 10 per cent of his series of 82 cases were chauffeurs. In Shawan's[591] series 5.7 per cent were chauffeurs. In our series only 5 (2.4 per cent) were chauffeurs. In a collected series of 1,467[105, 192, 355, 474, 540, 591, 640, 643, 677, 711] it was found that 59 per cent were classified as laborers and 18.2 per cent as having sedentary occupations. Of interest also is the fact that 2.7 per cent of this group had domestic or household duties.

Etiology.—Etiologic Considerations: An intelligent consideration of the causative factors of acute perforated ulcer must necessarily preclude a rational comprehension of the etiology and pathogenesis of gastroduodenal ulceration. Undoubtedly here as in peptic ulceration in general no single factor can be held solely responsible for their development. It is considered inopportune as well as inexpedient to attempt here a detailed exposition of the voluminous clinical, statistical, and experimental investigations that have been contributed to the bewildering pathogenesis of this disease. The reader interested in this phase of the subject is referred to previous publications.[144, 145] However, it is considered desirable to review briefly those factors which may be influential in the development of the peptic ulceration and the complication of perforation. In previous publications[144, 467, 471] there was presented a rational understanding of the physiologic principles involved in the development and maintenance of peptic ulceration. According to this concept ulcer is established by the summation of two groups of factors: (1) the predisposing, which are inherent and therefore uncontrollable; and (2) the precipitating, which are controllable. The former group is composed of: (a) tissue susceptibility, which may be defined as an inherent vulnerability of certain portions of the gastrointestinal tract such as the lesser curvature of the stomach, "magenstrasse," the duodenal cap, etc.; and (b) constitutional, which is difficult to define and must be considered as an ingrained susceptibility of certain individuals to gastrointestinal ulceration. Whereas, the former inherent characteristic is present in all individuals, the latter exists only in those individuals who develop ulcer in the presence of the precipitating factors. Thus, both these predisposing factors, that is, tissue susceptibility and constitutional predisposition,

may be present in an individual without peptic ulcer necessarily developing. However, ulceration does occur if to this predisposing group there are added the precipitating factors which consist of: (a) chemical, (b) mechanical or traumatic, and (c) infective.

These factors obviously play significant roles in the development of a perforated ulcer. It is interesting to speculate upon the question: Why do ulcers perforate? Stefanelli[622] believes that there is a true disposition to perforation. As previously stated, approximately 10 to 12 per cent of all gastroduodenal ulcerations (nonmalignant) perforate. The concept stated above helps explain the development but does not adequately explain the occurrence of perforation. Certainly there are factors which operate in the development of perforation just as in the development of the ulcers and their presence in a patient with an ulcer will cause perforation. It is our belief that all patients with gastroduodenal ulceration are potential candidates for perforation. However, only those perforate which are exposed to certain precipitating "perforative" factors. These precipitating perforative factors may consist of trauma, infection, and a local lack of tissue resistance.

Of these factors trauma has been considered as one of the most obvious. Such trauma may be either internal or external. Externally applied trauma is also of interest from the standpoint of the workman's compensation. Numerous authors have directed attention to the fact that such trauma can be an immediate causative factor in the development of perforation. Weir[684] in 1900 directed attention to cases of perforated ulcer which followed externally applied trauma, and Murphy and Neff[448] in 1902 referred to a case observed by a French surgeon in which the perforated duodenal ulcer resulted apparently from the patient's lifting a heavy log. Peck[500] in 1907 reported a case which was believed to be due to lifting a heavy tank of gasoline. Moynihan[438] in 1910 reported a case of perforation which apparently occurred following a fall of a heavy barrel across the patient's abdomen. Since then a number of cases have been reported, either the result of strenuous physical exertion, coughing, sneezing, or a violent blow to the abdomen.[37, 52, 87, 286, 387, 410] Powers[525] in 1925 reported 5 cases of duodenal ulcer which perforated following violent blows to the abdomen, and Morrow[433] in 1935 reported 5 similar cases. Physical effort as a traumatic factor has also been reported by numerous observers. However, the incidence of trauma as an exciting factor in perforations is not high; as a matter of fact, according to reported statistics, it occurs relatively rarely.[462] In the 51 cases reviewed by Weir[684] there were only 4 in which trauma was considered an immediate causative factor. Siroktine[608] found the incidence to be approximately 20 per cent in 51 cases. Meeker[410] found it to be 11 per cent in 27 cases; Meyer and Brams,[414] 3.2 per cent in 62 cases; Gerhardt,[238]

9 per cent in 105 cases; Beams,[37] 10.5 per cent in 76 cases; Fallis,[192] 2 per cent in 100 cases; Shawan,[593] 2 per cent of 389 cases; and Herzberg,[286] 1.9 per cent in 162 cases. In a collected series[37, 179, 192, 286, 410, 414, 551, 593, 608] of 1,260 cases there were 50 (4 per cent) in which perforation was believed to be due to trauma. From these statistical observations it would appear that externally applied trauma plays a relatively insignificant role in the causation of perforation. Corlette[123] studied this problem on the basis of laws of physics and showed that according to Boyle's law no increase of general intra-abdominal pressure can cause a strain in an ulcer and that perforation from such a cause cannot occur. However, contraction of the muscular walls of the stomach will increase the hydrostatic pressure within the organ without increasing the general intra-abdominal pressure and, if the difference in pressure passes the bursting strain of an ulcer, perforation will occur. Of further interest in this regard are the experimental observations made by Beams,[37] who found that physical exertion did not increase the motility of the stomach and in some cases actually caused diminution in the tone and activity of this organ.

Internal trauma has also been considered as an excitative factor of perforation. Of this type, roentgenologic examination with a barium meal has been one of the most frequently considered. A number of these cases have been reported.[8, 90, 178, 229, 354, 397, 482, 648] The frequency of perforation following x-ray examination with a contrast meal has been variously estimated. Himmelmann[289] and Panek[484] reported high incidences of 29.4 per cent and 22 per cent respectively, and Heim[277] and Herzberg[286] reported low incidences of 0.7 per cent and 0.6 per cent respectively. In a collected series of 1,672 perforations[192, 225, 271, 277, 286, 289, 333, 484, 551, 602, 603] there were 34 (2 per cent) cases which followed barium meal examination. Eckman[174] found that in a series of 522 ulcer patients examined roentgenologically there were only 5 perforations, an incidence of less than 1 per cent. Singer[602, 603] in 1934 stated that in more than 5,000 peptic ulcers examined radiographically there were only 4 perforations, an incidence of 0.08 per cent. Thus it would appear that barium meal examination is not a significant causative factor in perforation. Eckman[174] directed attention to certain symptoms and signs which contraindicate this type of examination of gastroduodenal ulceration. These were: recent bleeding, increasing distress, tenderness and epigastric rigidity, failure to obtain relief with food as promptly as previously, and excessive spasm. Singer[602, 603] believes that the status of the lesion, i.e., whether it is progressive or retrogressive, is of significance in determining perforation during roentgen examination and that for this reason such an examination should be postponed in patients with history of recent intensification of symptoms.

Another type of internal trauma which apparently plays some role in the causation of perforated ulcer consists of intragastric foreign bodies, such as bezoars and concretions. A number of observers have reported the occurrence of acute perforated ulcer in the presence of phytobezoars and trichobezoars.[47, 94, 171, 262, 288, 447, 529, 548, 556, 610] Ochsner and I[146] have made a comprehensive survey of the literature of this subject. It was found that of 250 collected cases of trichobezoars and phytobezoars gastroduodenal ulceration was present in 15.2 per cent. It was also found that of 15 collected cases of trichobezoars with ulcers, 5 (33.3 per cent) perforated; and of 19 collected cases of phytobezoars with ulcers, 7 (36.8 per cent) perforated. Thus, it is evident that the presence of an intragastric foreign body over a long period of time apparently not only predisposes to the development of ulceration but also increases the likelihood of perforation. This is shown by the statistics which reveal that in 34 cases of bezoars complicated with ulcers, 35 per cent perforated, an incidence considerably higher than that of perforation in gastroduodenal ulceration in general.

Obviously, one of the greatest sources of internal trauma is food and particularly indiscretions in diet. Pariser[488] in 1895 was impressed by the importance of presence or absence of food in the stomach determining the fate of a peptic ulcer. Noehren[462] was convinced that the most common immediate cause was the overloading of the stomach. Judine[323] was also of the opinion that perforation occurred most frequently after meals. However, a careful examination of his charts revealing postcibal perforation shows that the most frequent occurrence is during the third to the fifth hours inclusive, which places the perforation at least two hours following the meal. This is in agreement with the findings of others that the greatest incidence is at least two or more hours after meals. Meyer and Brams[414] found this to be true in about four-fifths of all their cases and stated that it was their belief that distention by food alone plays a comparatively minor role. Eliason and Ebeling[179] found that only 10 per cent of their cases perforated shortly after taking food, and Beams[37] records that in 60 (78.8 per cent) of 76 patients perforation occurred from two to four hours after the last meal. Chang[105] found that in his series of 35 cases the highest incidence was during the afternoon and in the morning. He also states that it is the custom in Peiping, where these cases were studied, for the majority of the poorer classes to take only two meals a day, at 10:00 A.M. and at 4:00 P.M. Accordingly, the highest incidence in this series occurred at least two hours after meals. Thus, it would appear from these statistics that distention of the stomach by food and by trauma of this type play relatively insignificant roles in the causation of perforation. Moreover, the fact that in a large percentage of cases perforation occurs while patients are at absolute rest, either actually asleep in bed or even in a hospital undergoing treatment for

peptic ulcer, seems to indicate that some other factors are significant.[54, 179, 213, 229, 483, 703]

Meyer and Brams[414] in 1926 reported that perforation occurred while patients were sleeping in 5 (8 per cent) of 62 cases. In Thompson's[640] large series 20.9 per cent perforated while asleep. Twigg[659] reported a case of a young aviator who developed an attack of perforation while flying at an altitude of 2000 feet. In Eliason and Ebeling's[179] 60 cases one-third perforated while they were asleep. Garver[229] reported 6 of 43 cases asleep at the time of perforation. Fallis[192] states that one-third of his series of 100 cases perforated while the patients were at rest. Assuming that in Russia people sleep between the hours of 11:00 P.M. and 7:00 A.M., in Judine's[323] series of 272 cases, 23.5 per cent perforated while patients were sleeping. Vale and Cameron[664] reported 5 cases of perforation occurring in patients in the hospital under conditions most favorable for healing; that is, while undergoing treatment for peptic ulcer. Of peculiar interest in this regard is that, paradoxically, these patients apparently had "less chance of recovery than the one who brings his perforation to the hospital." Dowdle[161] reported an unusual case in which a second ulcer perforated six days following the rupture and repair of the first one. From this it would appear that some other factor besides intragastric trauma plays an important role in the causation of perforation.

The influence of alcohol and tobacco in predisposing to perforation has been emphasized. Walker[674] found that two-thirds of 78 cases and Wolff[701] observed that almost four-fifths of 55 cases gave a history of alcohol imbibition. Lang[355] and Heim[277] also found a high incidence in their cases. Hull,[303] Fallis,[192] and Beams[37] observed lower incidence. Lewis[362] reported 3 cases which followed overindulgence in alcohol. Henle[280] believes that the recent increased incidence is due to abuse of alcohol. Undoubtedly alcohol plays a significant role in some cases and must be considered as one of the precipitating perforative factors. Similarly tobacco smoking as previously emphasized is also one of these factors.

Pathology.—Location: Considerable diversity of opinion seems to exist regarding the frequency of the site of perforation. There are several obvious reasons for this disagreement. At the time of operation, it is frequently difficult to determine the exact site of the perforation because of the distortion of normal anatomic relationship by the surrounding inflammatory reaction, fibrinous adhesions, and peritoneal exudate. Moreover, the surgeon is usually reluctant to disturb the surrounding structures by traumatic manipulation and traction in an attempt merely to ascertain whether the perforation is in the prepyloric or duodenal segments. In addition to these difficulties, there is ap-

parently some discrepancy among various authors as to whether the pylorus is part of the stomach or of the duodenum.

In view of these facts it is not surprising to see such great variations among reporters of the frequency of gastric and duodenal perforated ulcers. Thus, Bager[21] in a series of 1,488 cases found that

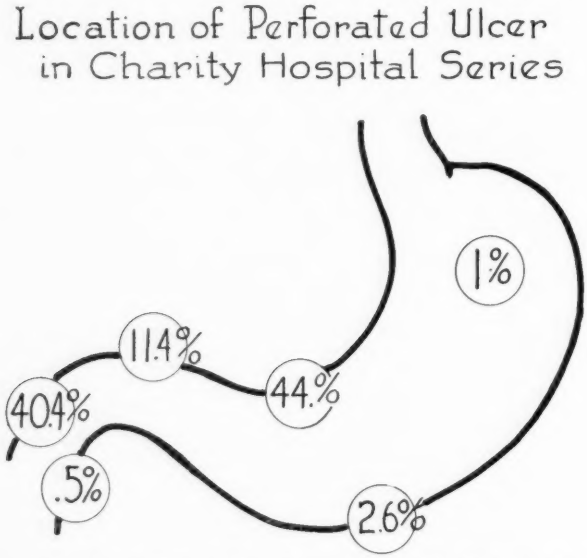
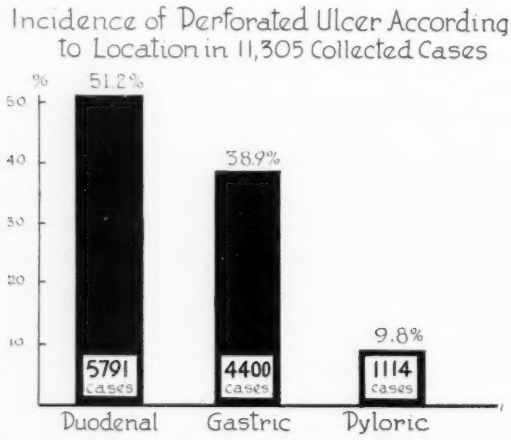


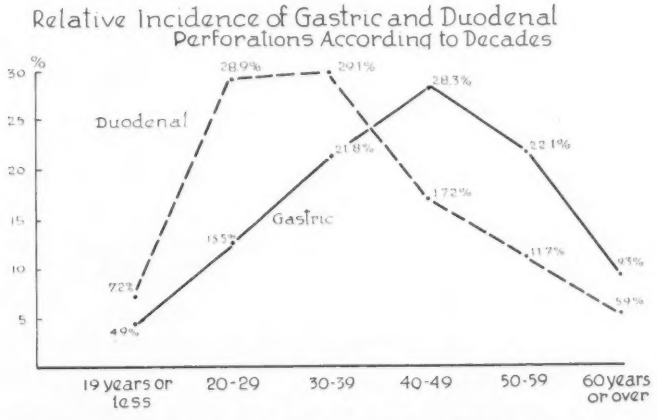
Fig. 1.—Diagrammatic representation of comparative incidences of site of perforation as based upon 211 cases in the Charity Hospital series. It may be observed that the large majority of ulcers are juxtapyloric.



Graph VIII.—Comparative incidences of site of perforation in gastroduodenal ulceration.

69.3 per cent were gastric and Heim[277] in a series of 283 cases of perforated ulcer stated that 75.6 per cent were gastric. Somewhat similar figures were given by Chiasserini,[109] Delrez and Bottin,[150] Loehr,[368] and Wilmoth.[696] Numerous others state that gastric ulcers were more common.[187, 229, 403, 454, 560, 674, 677] On the other hand, Moynihan,[441]

White and Patterson,[688] Hagyard,[267] Gilmour and Saint,[243] and Judine[323] found in relatively large series that perforations of the duodenum occurred in approximately 90 per cent or over. Many other observers agree that duodenal ulcer perforations are more frequent.[29, 82, 87, 136, 181, 238, 593] Judine[323] is quite decided in his conviction that 90 per cent of perforations are in the duodenal segment. He states that in his series of 928 perforated ulcers 812 (87.5 per cent) were on the duodenal side. His figures are based upon careful examination of resected gastroduodenal segments and microscopic differentiation of the respective mucosae. In the Charity Hospital series of 193 cases in which the site of perforation was stated, 92 (47.6 per cent) were gastric and 101 (52.3 per cent) were duodenal (Fig. 1). In a collected series of 11,305 cases it was found that 4,400 (38.9 per cent) were classified as gastric, 5,791 (51.2 per cent) as duodenal, and 1,114 (9.8 per cent) as pyloric[718] (Graph VIII).



Graph IX.—Relative incidences of gastric and duodenal ulcer perforations according to decades. It will be observed that, whereas the highest incidence of duodenal ulceration occurs in the third and fourth decades, in gastric ulcer perforations the highest incidence occurs in the fourth through the sixth decades.

Of particular interest in this regard is the factor of age incidence. It would appear that the average age of perforated duodenal ulcer is approximately a decade less than that of perforated gastric ulcer. Judine [323] found that the average ages in his series were 32 years for duodenal ulcer perforations and 46 years for gastric. This greater frequency of perforated gastric ulcer in elderly patients has been observed also in our cases. In a collected series of 2,429 cases,[21, 323, 420, 474, 535] including ours in which estimates of this nature could be made, it was found that, whereas 65 per cent of the perforated duodenal ulcers occurred in patients under 40 years of age, 60 per cent of the perforated gastric ulcers occurred in patients over 40 years of age (Graph IX). It would appear, therefore, that perforated duodenal ulcers are more likely to occur in younger patients and gastric ulcers in older patients.

With rare exceptions perforated ulcers occur on the anterior aspect of the stomach and duodenum. In a series of 343 cases of perforated gastric ulcer, Radoievitch[530] found that only 7 (2.0 per cent) were located posteriorly and in 259 perforated duodenal ulcers only 5 (1.9 per cent) were posterior. A much higher incidence was reported by Eliason and Ebeling,[179] who found 2 (14.3 per cent) posteriorly located perforations in 14 gastric ulcers and 2 (5.4 per cent) in 34 duodenal ulcers. Thompson[640] found 71.4 per cent of 483 cases located anteriorly. In the Charity Hospital series[474] of 92 cases of gastric perforations 4 (4.3 per cent) were in the posterior wall and in 101 duodenal ulcers 2 (1.9 per cent) were posterior. In a collected series of 1,523 perforated ulcers only 33 (2.16 per cent) were located posteriorly.[179, 192, 229, 238, 330, 355, 402, 514, 640, 643] The obvious explanation for the rarity of posterior perforations lies in the fact that the posterior aspect of the stomach and duodenum is more intimate with the adjacent structures; whereas, the anterior wall is more readily exposed to the general peritoneal cavity.

Multiple perforated gastroduodenal ulceration occurs relatively rarely. This is shown clearly by reported statistics. Radoievitch[530] in an analysis of 718 collected cases stated that he had found only 3 with multiple perforations. Judine,[323] whose experience included approximately 900 cases of perforated ulcer, stated that he has observed multiple perforations only once, although multiple ulcers were present in 8 cases. Among the first to report the occurrence of multiple perforations were Bott[64] in 1876, Vergely[669] in 1881, and Mikulicz,[418] who in 1889 reported a case which he treated surgically. Brentano[75] in 1906 found 5 multiple perforations in 75 cases. In 1909 Eliot[182] was able to collect 18 cases of multiple perforations. Finney[203] in 1900 stated that "statistics show that in 20 per cent of cases there is more than one perforation." This incidence seems unusually high. Brunner[84] believes that from one-fourth to one-third of all ulcers are multiple. Judd and Proctor[318] believe that multiple ulcers occur in 6 per cent of cases. Hurst and Stewart[306] found that the incidence of multiple ulcers in post-mortem cases was 12.2 per cent and in surgically excised stomachs 8.5 per cent. However, the incidence of multiple perforations is lower. Villard and Pinnatelle[672] reported an incidence of 3.5 per cent in 115 cases and Petrén[507] found the incidence to be 4.3 per cent in 92 cases. In 1914 Caird and associates[96] reported an incidence of 0.8 per cent in a series of 247 cases. In 1927 Masson and Simon[393] reported a case of multiple perforation and in a review of the literature stated that they were able to find only 32 authentic cases. A number of cases have since been reported.[18, 165, 222, 609] In a collected series of 879 perforated ulcers there were 36 (4.1 per cent) with multiple perforations[228, 333, 355, 432, 528, 532, 551] In the majority of reported cases only 2 perforated ulcers existed. However, Vianny[670] and Brentano[75] reported cases in which

there were 3 and 4 perforated ulcers respectively. Usually the perforations are of the kissing ulcer type, being opposite each other on the anterior and posterior walls of the stomach. Occasionally, however, they are on the same side and rarely there is a perforated ulcer in the stomach and one in the duodenum. They are usually located on the gastric side near the pylorus and lesser curvature.

Perforations Into Other Organs.—Perforations of gastroduodenal ulceration occur occasionally into adjacent viscera rather than into the peritoneal cavity. This, however, is a relatively rare occurrence. It is apparently dependent upon the adherence of the ulcer area by peritoneal adhesions to these structures. Cases have been reported in which the perforation extended into the gall bladder,[217] pancreas,[101, 185, 497] spleen,[422] lung,[589] diaphragm,[301] extrahepatic bile ducts,[33] and gastrohepatic ligament.[381]

Gross Appearance.—The appearance of the lesion is usually quite characteristic. The perforative opening is almost invariably found near the pylorus in the prepyloric area, close to the lesser curvature or in the suprapapillary portion of the duodenum. The opening is most commonly single, round, and from a few millimeters to approximately a centimeter in diameter. Varying degrees of induration may be found in the gastroduodenal wall surrounding the opening. Brenner[71-74] classifies perforated duodenal ulcers into two general groups: (1) the soft lesions which are the most common type, characterized by their smaller size, their superficial extent, and absence of dense induration and adhesions to neighboring structures; and (2) the callous type, characterized by their large size, deeper penetration, and the presence of induration and adhesions to neighboring structures. The serosal surface surrounding the perforation presents a dull granular appearance and a varying degree of peritoneal exudate, depending upon the time elapsing between the perforation and the operation. In the later stages there is frequently observed a purulent greenish viscid exudate; whereas, in the early stages this is more fibrinous in character. Similarly, the peritoneal fluid also varies in appearance and may be either slightly turbid, fibrinous, or actually purulent. In some instances small food particles may also be seen in the neighborhood of the perforation.

Bacteriologic Considerations.—It has long been recognized that peritonitis is one of the most important complications of perforated gastroduodenal ulcer. It is generally believed that in the early postperforative period a chemical peritonitis occurs as a result of peritoneal contamination of the extruded acid gastric chyme and duodenal juices and that subsequently a bacterial peritonitis develops. Whereas the former type of peritonitis is considered relatively innocuous, the latter is undoubtedly serious.

There is a relative paucity of literature on bacteriologic studies of the peritoneal exudate in perforated ulcer. Among the earliest to make

such investigations were Dudgeon and Sargent,[167] who in 1905 were able to isolate gram-negative streptodiplococci from the edge of gastric ulcer in all and from the peritoneal exudate in 4 of 7 cases. Of 4 duodenal ulcer cases the cultures were sterile in 3 and showed *Bacillus coli* in 1. Two of the gastric ulcer cases and 2 of the duodenal ulcer cases died. The authors were impressed with the low pathogenicity of the isolated diplostreptococcus. In a subsequent publication[166] in which the results of 23 cases were reported, 13 were found sterile and 10 showed diplococci and streptococci. They directed attention again to the nonhemolytic and low pathogenetic characteristics of these organisms. It was also observed that colon bacilli were found in the only 2 fatal cases. Deaver[140] in 1913 found that of 20 cases in which cultural studies were made, 14 showed no growth, 4 showed *B. coli*, 1 revealed streptococci, and 1 staphylococci. Walker[674] in 1915 found negative cultures in 14 of 29 and subsequently reported sterile cultures in 20 of 34 cases. A rather high incidence of sterile cultures was reported by Richardson,[545] who found 28 of 35 cases negative. An even higher incidence was reported by Brenner,[74] who found all of 21 cases negative. In Deaver and Pfieffer's series[142] 23 of 34 cases were sterile and in Graham's[248] 30 cases 20 were sterile. Vendt[668] found positive growths in 77 per cent of 16 cases with nonhemolytic streptococci as the predominant organism. Fallis[192] reported 12 positive cultures in 36 cases and McCreery[403] reported 22 positive cultures in 42 cases. The streptococci and staphylococci were the predominant organisms and *B. coli* was found in the late cases. Chang[105] reported 13 positive cultures in 31 cases, with the streptococcus as the predominant organism.

Obviously, the presence or absence and the incidence of positive cultures will depend upon the length of time elapsing between the perforation and the performance of the culture. The cultures are more likely to be positive, the greater is this period. Whereas, some observers[6, 438, 660] have maintained that the peritoneal fluid is sterile up to twelve or eighteen hours, others[85, 323] have reported high incidences of positive culture made in less than twelve hours. Brütt[85] performed cultures in 112 cases and found growths in 74 per cent of those operated upon six to twelve hours following perforation and in 93 per cent of those operated upon twelve hours or more after perforation. Judine[320] in 100 cases in which culture studies were done found positive growth in 89 per cent of those examined within twelve hours after the perforation and in 100 per cent after twelve hours. The predominant organisms were streptococci and staphylococci. Davison and co-workers[138] in 34 cases studied found 18 per cent positive cultures in the first six hours, and 75 per cent positive cultures in the second six hours. The significance of anaerobic bacteria has been studied by Lohr[369] and Meleney and associates.[411, 412]

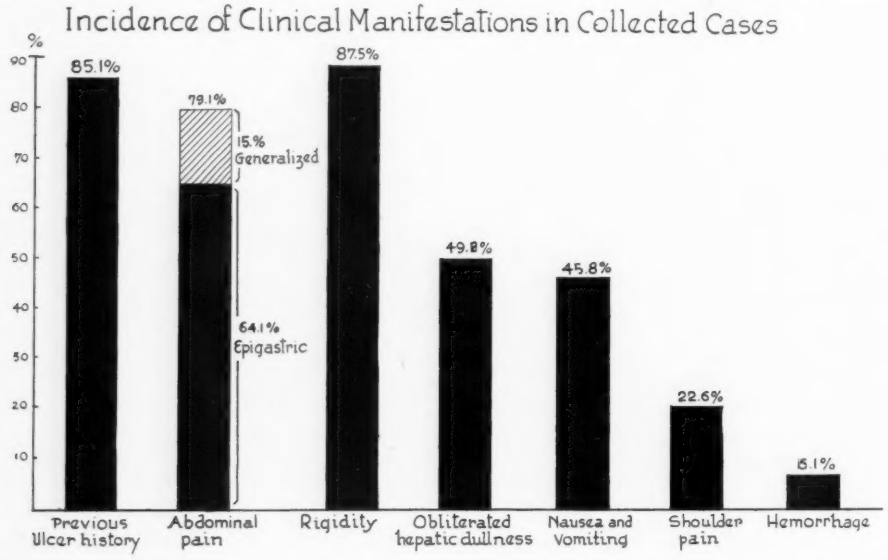
Undoubtedly the presence of hydrochloric acid produces an inhibitory effect on the bacterial growth. This was very early shown by the extensive investigations of Cushing and Livingwood,[132] who found that there is a "tendency on the part of the stomach to completely free itself —from even those microorganisms which are resistant to the gastric juice." Numerous other investigators[12, 34, 224, 314, 334, 345, 452, 479, 520, 543, 565, 634, 663] have since directed attention to this fact. It would appear, therefore, that the acid gastric juice has a bacteriostatic effect upon microorganisms. Thus, in the early stages of the perforation the bacteriostatic effect of the hydrochloric acid is in operation and relatively fewer organisms will be found during this period. During the later stages, this inhibitory effect is no longer operative and the bacterial flora changes so that streptococci and *B. coli* become more prominent. Davison and associates,[138] who have directed attention to this fact, explain it on the basis that there occurs "a cessation of peristalsis with a concomitant cessation of secretion of hydrochloric acid" consequent to the early chemical peritonitis which permits proximal migration of bacteria from the lower bowel. Thus the greater is the postperforative period, the more likely is the development of pathogenic bacteria. Obviously, therefore, the bacterial flora has an important prognostic influence. Thus, in Fallis'[192] series the mortality in those cases with a positive culture was double the mortality for the entire series. In Judine's[323] series none of the cases with sterile culture died. Clavel and Clavel[114] and Michon[416] found that only 1 of 7 cases with sterile cultures died; whereas, 3 of the 6 cases with positive cultures died. Davison and associates[138] observed that, although the mortality in 8 cases with positive cultures was 66.6 per cent, in the 16 cases with sterile cultures it was 25 per cent. On the basis of their experimental observations Bergh, Bower, and Wangensteen[42] classified the factors influencing the development of peritonitis, and consequently the prognosis, into two general groups: (1) the number and virulence of the escaping organisms, and (2) the resistance of the host.

Trout[652] directed attention to the fact that a study of the bacterial flora of the mouth, teeth, and throat should be made because the organisms found in the stomach will depend largely on the bacteria found in these regions. Such a study has been reported by Arapov and Grasse,[11] who found that there were three principal groups of organisms: (1) the ordinary saprophytes; (2) the cocci, including diplococcus, streptococcus, diplostreptococcus, and saprophytes; and (3) the spirochetes. Moreover, they found that of this group the microorganism most resistant to antiseptic mouthwashes, including 1 per cent hydrochloric acid, was the diplostreptococcus. This is of interest because Judine[323] observed that in the early hours after perforation the peritoneal fluid contains a high incidence of microorganisms normally found in the oral cavity; whereas, in the later stages the incidence of the diplostrepto-

coccus increased. On this basis oral hygiene would seem to have little influence upon the development of postperforative peritonitis.

Schmeil[569] performed serial sections and histologic studies on 50 resected stomachs for perforative ulcer in an attempt to determine the significance of the bacterial flora. At the mucosal surface of the ulcer and in the tissue surrounding the perforation, the bacterial flora was found to be similar in 94 per cent of the cases. He concluded that bacteria play an important role in the development of the perforation.

Clinical Manifestations.—The clinical manifestations of perforated gastroduodenal ulceration are quite characteristic and have been described adequately in previous publications.[120, 278, 438, 440-442, 485, 486, 498, 604] Whereas no attempt will be made here to present a detailed description of these characteristic symptoms and signs, it is considered desirable to direct attention to some of the more prominent features. Clinically, three stages of the disease are recognized: (1) the primary stage, in which the dramatic symptoms reflecting an acute peritoneal reaction



Graph X.—Respective incidences of clinical manifestations in gastroduodenal ulceration.

are believed to be the result of extravasation of acid chyme and duodenal juices into the peritoneal cavity; (2) an intermediate stage, which represents the recovery from the primary stage and in which the patient's condition appears somewhat improved; and (3) the tertiary stage, in which the manifestations are characteristic of bacterial peritonitis.

At the onset of the perforation, the patient's most prominent complaint is severe agonizing pain, usually localized to the epigastrium. Whereas, this pain varies in intensity, it is most commonly considered as one of the most severe and agonizing types of pain that it is possible

to experience. Although in some patients there are prodromal manifestations and the pain gradually becomes more severe, in the majority the pain strikes suddenly and with maximum intensity. Numerous adjectives, such as intolerable, agonizing, violent, tearing, racking, bursting, horrible, knifelike, stabbing, burning, etc., have been used to describe the pain but its most characteristic features are its intensity and constancy. Whereas, sometimes the pain is generalized over the entire abdomen, it is most frequently located in the epigastrium and occasionally to the right of the midline. It may also be referred to the left upper quadrant, the right lower quadrant, the back, or the shoulders. In the Charity Hospital series of cases the pain was generalized in 60 (28.4 per cent), localized to the epigastrium in 115 (54.5 per cent) and to the right lower quadrant in 6 (2.8 per cent). In a collected series of 2,132 cases[719] the pain was found to be generalized in the abdomen in 321 (15 per cent) and localized to the epigastrium in 1,367 (64.1 per cent), to the right upper quadrant in 59 (2.8 per cent), to the right lower quadrant in 105 (4.9 per cent), to the left upper quadrant in 7 (0.3 per cent), to the back in 34 (1.6 per cent) (Graph X). Pain referred to the shoulders is an interesting phenomenon. Several observers[240, 476] have directed attention to its occurrence and Caird and co-workers[96] in 1913 stated that it was present in 7 of 100 cases. Whereas, White and Patterson[688] observed it in none of their 62 cases, Bryce[87] found it present in 74 (68.5 per cent) of 108 cases. In the Charity Hospital series of 211 cases it was found in 32 (15.1 per cent). In a collected series of 1,657 cases[720] shoulder pain was found present in 375 (22.6 per cent) (Graph X).

The actual cause of the pain in perforated gastroduodenal ulceration has been a question for considerable discussion. Moynihan[442] has expressed the opinion that the early symptoms and signs are due to disturbed reflexes. The viscera are associated through their sympathetic nerves with the lower six dorsal nerves and an irritative lesion of a viscus produces a viscerosensory reflex by transmission of the stimulus to the corresponding spinal segment with resulting expression of pain or hyperalgesia. That the intense pain may be due to a reflex pylorospasm has been observed by Paas[482] in perforations of the duodenum in basis of experimental observations in dogs, believes that pylorospasm may be produced by an irritation of the peritoneal surface. Such a spasm has been observed by Pass[482] in perforations of the duodenum in dogs and by Watson[679] in human beings. Mackenzie[379] early expressed the belief that violent contraction of the smooth muscle of a hollow viscus played a significant role in the production of visceral pain. It would appear, therefore, that the intense pain experienced in acute perforated ulcer is the result of the profound peritoneal irritation by contact with the gastroduodenal contents, producing violent pylorospasm

and setting up an irritable focus in the lower dorsal spinal segments by stimuli transmitted over sympathetic nerves.

In addition to the intense pain the next most characteristic clinical feature of this early stage of perforation is prostration. Some confusion has existed regarding the term applied to this particular phenomenon. Because characteristically in this early stage the patient's appearance reveals an anxious, pale, and haggard expression, the brow and temples bathed in perspiration, the mouth tightly closed, the nostrils wide, the respirations shallow, costal, and panting in character, the cutaneous surface rather cold and clammy, and the body held rigid and immobile, the term "shock" has been applied to describe the condition. Actually, however, as emphasized by Moynihan these patients are not in true surgical shock because the pulse rate is usually not increased and the blood pressure is rarely diminished. For this reason, Moynihan suggests "prostration" as a better term. Eliason and Ebeling[179] observed only 4 of 46 cases with blood pressures less than 100 mg. mercury. In 63 cases that recovered McCreery[403] found that the systolic blood pressure was less than 100 in only 1; whereas, in 19 fatal cases it was less than 100 in 7. Thompson[639] found the blood pressure normal in 80 per cent of 364 cases. A number of observers[73, 74, 192, 230, 403, 550, 592, 593, 679, 688] have directed attention to the fact that this so-called shock has been overemphasized. Moreover, when such shock does exist, it is a grave sign.

These early severe manifestations of acute perforated ulcer immediately raise the question of mechanism. Obviously, infection cannot be considered a factor because the manifestations occur before infection can develop. In this regard the experimental observations of Blalock[56] are particularly interesting. The upper intestinal juices, bile, pancreatic juice, duodenal secretions, and gastric juice were separately collected from dogs and injected singly or in various combinations into the peritoneal cavities of other dogs. The symptoms observed in these animals following the intraperitoneal injection of the various secretions were similar to those seen in human beings following acute perforated ulcer. Studies on cardiac output and blood pressure revealed a decline in blood pressure and subsequent greater drop in cardiac output. Whereas, bile was found to exert a more deleterious effect than any one of the other secretions, a mixture of bile and pancreatic juice produced the most marked changes. Blalock[56] believes that the early manifestations are not due to absorption of toxic material but rather to the chemical irritant action of the juices. Based upon these studies his conception of the alterations in circulation following the intraperitoneal injection of these secretions is as follows: "A dilatation of many small vessels is produced by direct contact with the irritating solutions, and to a lesser extent, through nervous agencies. This results in a drop in the blood pressure. After the blood volume de-

creases as a result of the marked increase in the circulatory bed and the passage of fluid into the peritoneal cavity, the output of the heart diminishes. The blood pressure is probably prevented temporarily from declining further by vasoconstriction elsewhere.''

The occurrence of nausea and vomiting is inconstant and its frequency in reported statistics varies from the low incidence of 13 per cent[542] to the high incidence of 95.8 per cent.[188] In the Charity Hospital series of 211 cases nausea and vomiting were present in 87 (41.2 per cent). In a collected series of 2,190 cases[721] nausea and vomiting were found present in 1,003 (45.8 per cent) (Graph X).

Of the physical findings in acute perforated ulcer, abdominal tenderness and rigidity are probably the most constant and prominent. Tenderness is usually most marked in the upper abdomen. In describing the characteristic rigidity, Moynihan[442] states: ''Every part offers the most inflexible opposition to pressure; the rigidity is obdurate, persistent, and unyielding.'' The significance of this rigidity which is frequently spoken of as ''boardlike'' as a diagnostic sign of acute perforated ulcer is shown by the fact that in a collected series of 2,011 cases[722] it was present in 1,760 (87.5 per cent) (Graph X). In 202 cases of the Charity Hospital series in which these facts were recorded 187 (92.6 per cent) had such rigidity.

Obliteration of hepatic dullness is a variable finding. Some observers[255, 618] stress its importance in the diagnosis of acute perforated ulcer; whereas, others[329] believe it to be relatively insignificant. Field[201] in 1918 suggested that by changing the position of the patient its incidence of occurrence could be increased. Its incidence varies in the reported statistics from 0 per cent by Kellogg[329] to 100 per cent by Soupault[618] and Chabrut.[104] In a collected series of 1,133 cases[723] including my own cases it was found present in 558 (49.2 per cent) (Graph X). Obviously its presence should be considered a confirmatory sign, but its absence should not be considered refutatory.

The temperature and pulse rate in the early stage of perforation are usually not elevated or, if elevated, are not very high. Bryce[87] in a series of 129 cases found the average pulse rate to be between 60 and 80 per minute. Others[22, 74, 117, 432] have also found both the temperature and pulse rates practically normal. Thompson[639] in a series of 469 cases found the temperature normal in 68.6 per cent and the pulse rate normal in 74.8 per cent. In fact, an actual slowing of the pulse rate has been observed and Kreeke[342] explains this on the basis of reflex vagal stimulation. In the later stages of perforation both the temperature and the pulse rate become definitely increased. The respirations, even in the early stage, are usually rapid. Brenner found the average respiratory rate 23 per minute; Read,[540] 24; Robitshek[550] and Turberville,[656] 28; and Deaver and Pfieffer,[142] 31. In the 469 cases examined by Thompson[637] the respiratory rate was increased in 62.3 per cent.

Leucocytosis is an early finding in acute perforated ulcer. Robitshek,[550] Colp,[117] Read,[540] Deaver and Pfieffer,[142] Brenner,[74] and Donald[158] found the average leucocyte count to be about 15,000 with a corresponding increase in the polymorphonuclear leucocytes. Fallis[192] found that the leucocyte count was under 10,000 in only 11 per cent of 81 cases and over 15,000 in 48.2 per cent. The polymorphonuclear leucocytes were over 80 per cent in 77.5 per cent of the patients. Shawan[593] reported an average leucocyte count of 13,385 in a series of 183 cases and an average polymorphonuclear count of 81 per cent. Ross and Letourneau[551] found that the leucocyte count was between 10,000 and 18,000 in 70 per cent and over this in 16 per cent. In the Charity Hospital series of cases the highest leucocyte count was 36,500, the lowest 3,800, and the average 14,114.

Hemorrhage in association with perforation is usually considered as a rare occurrence. However, the coexistence of these two serious complications has been recorded.[41, 198, 538] The frequency of hemorrhage in peptic ulcer has been emphasized by Bevan[48] and Balfour,[25] although Behrend[38] believes that perforations occur in a higher incidence. The statement that "bleeding ulcers rarely perforate and perforating ulcers rarely bleed" is frequently observed in the literature. The incidence in the reported statistics varies considerably but rarely exceeds 10 per cent. In a collected series of 2,525 cases of perforated ulcers[724] an associated hemorrhage was found in only 155 (6.1 per cent) (Graph X). In the Charity Hospital series of 211 cases this incidence was only 1.9 per cent. This question of associated perforation and hemorrhage has recently been reviewed by Winters and Egan.[699] These observers found that of 13 patients coming to autopsy who had perforation without bleeding 3 had multiple ulcers; whereas, of 8 patients who had perforation with bleeding, 7 had multiple ulcers. Of their 152 gastric perforations, 16 (10.5 per cent) had accompanying hemorrhage. Probably the reason for the relative infrequency of hemorrhage in perforated ulcer lies in the peculiar vascular distribution of that gastroduodenal area in which perforation is most common.[177, 541, 692]

Within several hours after the onset of the perforation the severe dramatic manifestations begin to subside, the patient's condition seems to improve, and gradually there appears the reaction stage or so-called stage of delusion. The pain subsides somewhat, the body surface seems warmer, and the expression of agony and anxiety seems less intense. However, abdominal tenderness and rigidity are still prominent, respiration remains costal and rapid, and, if the condition is allowed to progress without surgical intervention, the patient soon passes over into the third stage or stage of generalized peritonitis. The temperature and pulse rate begin to rise, abdominal tenderness and rigidity become less marked; whereas, ileus becomes prominent, singultus may be observed, and the characteristic features of peritonitis become obvious.

Diagnosis.—The diagnosis of acute perforated gastroduodenal ulceration is usually not difficult if the condition is kept in mind and careful consideration accorded the characteristic clinical manifestations. Whereas in some cases a diagnosis cannot be definitely established, in the majority of instances not only can it be determined that the abdominal condition is one requiring immediate surgical intervention, but frequently it can be ascertained that it is due to an acute perforated ulcer. However, as emphasized by Moynihan,[442] although it is desirable to make an accurate diagnosis, "it is important only to know that something has happened with the abdomen which cannot be relieved by any other than surgical measures." The clinical picture of an acute abdominal catastrophe may be reflected by a number of pathologic conditions other than acute perforated ulcer. The determination of the exact cause of the calamity is optative, but the recognition of a surgical exigency is imperative. In this regard the words of Moynihan are apt: "To make an accurate anatomical and pathological diagnosis is a delight; to reach a decision that, wherever and whatever the trouble, the sooner measures of rescue are adopted, is a cardinal obligations."

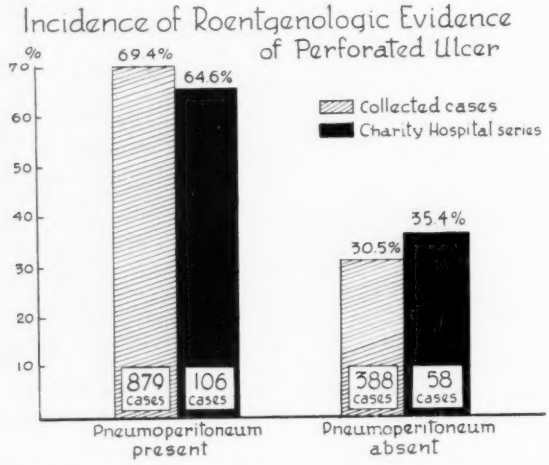
In the majority of cases recognition of an abdominal catastrophe is relatively simple. The severe agonizing abdominal pain usually localized to the epigastrium, the prostration in the early cases, the minimal degree of nausea and vomiting, and the marked tenderness and rigidity are clinical features characteristic of an acute surgical abdomen and highly suggestive of acute perforated gastroduodenal ulceration. Obliteration of hepatic dullness and in the early stages the low temperature and pulse rate are additional evidences.

The history of previous gastric disturbances suggestive of gastroduodenal ulceration is further proof and can usually be obtained by closely questioning the patient. The majority of patients will give such a history with duration of months or years. In Thompson's[639] series of 303 cases 14.5 per cent gave a history of previous disturbances for from one to twelve months, 41 per cent for from one to five years, and 36.9 per cent for over five years. In Graves'[253] 144 cases 90 per cent had such histories and Judine[323] found similar histories present in 95 per cent of his group. Ross and Letourneau[551] in a series of 213 cases reported previous ulcer histories of under six months in 23 per cent, of from six months to two years in 16.4 per cent, of from two to five years in 20.6 per cent, and of over five years in 31.1 per cent. In a collected series of 6,110 cases[725] previous ulcer history was present in 5,206 (85.1 per cent) (Graph X). In the Charity Hospital series of 211 cases 159 (75.3 per cent) were characterized by long histories of gastric disturbances and in 23 (11 per cent) the histories of gastric distress were over a relatively short period of time, usually several weeks.

It should be realized, however, that in some cases the perforation forms the first manifestation of gastroduodenal ulceration. This was

present in 2.6 per cent of Sherren's[595] cases, 9.7 per cent of Nielson's,[459] and 12.2 per cent of Brown's.[83] Others have reported even higher incidences. Thus, the perforation was the first manifestation in 23 per cent of Morrison's[432] series of 200 cases, in 21.3 per cent of Rhodes and Collins',[542] in 19.1 per cent of McCreery's[403] 170 cases, in 15.7 per cent of Semb's[588] 156 cases, and in 13 per cent of Shawan's[593] 389 cases. In the Charity Hospital series of 211 cases 29 (13.6 per cent) gave no previous ulcer history. In a collected series of 6,110 cases[725] 904 (14.8 per cent) had absence of previous ulcer history.

Undoubtedly one of the most reliable diagnostic procedures in acute perforated gastroduodenal ulceration is roentgenographic demonstration of spontaneous pneumoperitoneum. Although Weinberger[681] had observed radiologically the so-called "subphrenic pyopneumothorax" in 1908, this procedure was originally suggested in 1915 by Popper,[521] who four years previously observed a case in which air beneath the diaphragm was demonstrable upon fluoroscopic examination and was interpreted as having escaped from a perforated peptic ulcer. A somewhat similar observation was made by Wieland[690] in



Graph XI.—Incidence of roentgenologic evidence of perforated gastroduodenal ulceration.

1915. The following year Lenk[360] reported his roentgenographic observations of intraperitoneal free air in abdominal gunshot wounds and suggested the value of this examination in the differentiation of perforated ulcer from other conditions. Following these suggestions and during the next few years a number of solitary case reports indicating the diagnostic significance of this procedure appeared.[726] In 1925 Vaughan and Brams[665] reported 13 (86.7 per cent) of 15 cases of anatomically proved acute perforated gastroduodenal ulceration in which radiographic examination revealed free gas in the peritoneal cavity. In a subsequent publication this subject was carefully re-

viewed by Vaughan and Singer,[667] who reported fluoroscopically demonstrable free intraperitoneal air in 54 (85.7 per cent) of 63 definitely established cases of perforated ulcer. During the past decade a number of observers have emphasized its diagnostic value.[727] Positive results have been reported in incidence of 67.7 per cent,[323] 70 per cent,[515] 83.3 per cent,[313] 98 per cent,[337] and 100 per cent.[348] In the Charity Hospital series[474] of 164 cases in which this examination was made, 106 (64.6 per cent) showed air beneath the diaphragm. In a series of 1,267 cases collected from the literature[728] in which the results of this examination were stated, there were 879 (69.4 per cent) with positive evidence of pneumoperitoneum (Graph XI).

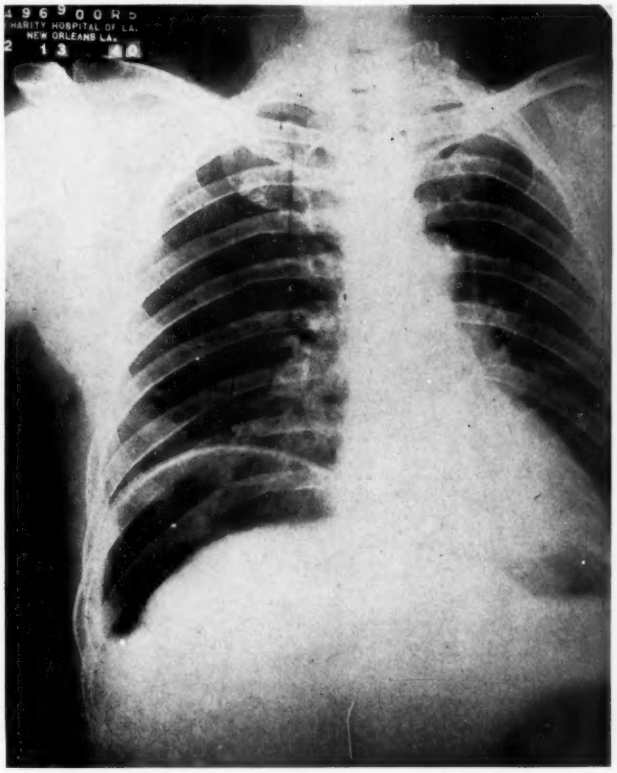


Fig. 2.—Anteroposterior roentgenogram taken in erect position of abdomen of patient with perforated ulcer showing somewhat crescent-shaped translucent area under right leaf of the diaphragm representing escaped gas.

The technique of performing this valuable diagnostic procedure should be accomplished with facility and with absence of discomfort or danger. By placing the patient in the sitting or upright position, gas is demonstrable upon fluoroscopic examination immediately beneath the right leaf of the diaphragm and between it and the upper surface of the liver. The translucent area representing the gas is characteristically somewhat crescent shaped with sharply demarcated boundaries (Figs. 2 and 3). Upon moving the patient in other posi-

tions the gas bubble moves to assume the highest level. Thus, by having the patient lie in the left lateral position, the air will be found under the right lateral abdominal wall and between it and the liver (Fig. 4). Fluoroscopic examination is sufficient although roentgenograms may be made if permanent records are desired.

In a previous publication[474] attention was directed to a possible means of increasing the incidence of positive roentgenographic findings. The procedure consists of introducing a stomach tube, aspirating the gastric contents, and injecting 20 to 30 c.c. of air into the stomach. The patient is then placed in the left lateral position and fluoroscopic examination made. After lying in the left lateral position for a few

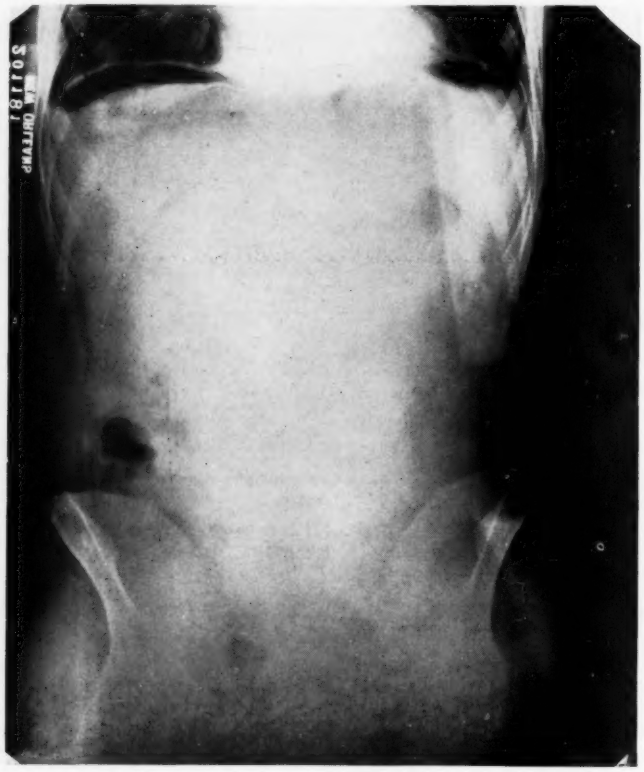


Fig. 3.—Anteroposterior roentgenogram taken in erect position of chest and abdomen of patient with perforated ulcer. The characteristic crescent-shaped translucent area between the right leaf of the diaphragm and the liver represents gas which has escaped from the perforation.

minutes, he may be placed in the sitting position and fluoroscopic examination will reveal air beneath the diaphragm. Obviously, this is not advocated as a routine procedure but only in those cases in which acute perforated ulcer is suspected and in which fluoroscopic examination does not reveal pneumoperitoneum.

Whereas, the fluoroscopic or roentgenographic demonstration of spontaneous pneumoperitoneum in a patient suspected of having acute

perforated ulcer may be considered for all practical purposes a pathognomonic sign, it should be realized that spontaneous pneumoperitoneum does occur in other conditions. Cases have been observed in which free intraperitoneal air was roentgenographically demonstrated following acute perforated tuberculous ulcer of the appendix[331] and the small bowel,[667] suppurative appendicitis,[260, 667] and typhoid perforation.[135, 667] Colson, Mansuy, and Caillard[118] reported a series of 59 cases of spontaneous pneumoperitoneum, only 21 of which were due to perforated ulcer. It should also be realized that the absence of demonstrable free intraperitoneal air in a case having other characteristic manifestations of acute perforated ulcer does not exclude the diagnosis.[522] However, these facts do not detract from the diagnostic value of the procedure. This is clearly demonstrated by the high incidence of positive findings in the collected cases, including the author's

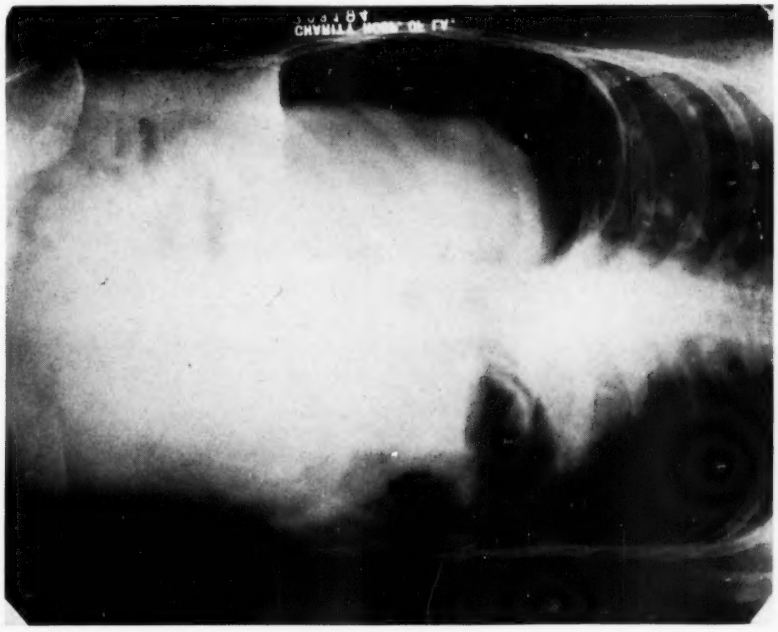


Fig. 4.—Anteroposterior roentgenogram taken in the left lateral position of abdomen of patient with perforated ulcer showing translucent area representing gas under the right lateral abdominal wall and between it and the liver.

(Graph XI). Every aid should be employed in an attempt to establish a definite diagnosis, and in perforated ulcer this is frequently difficult. Roentgenographic examination provides corroborative evidence which may be obtained simply and easily and which, if employed routinely, will undoubtedly increase the accuracy of diagnosis.

There have been several reports[17, 401, 517, 671] in the recent literature on the presence of subcutaneous emphysema as an unusual finding in perforated ulcer. Perforation into the pancreas was found to be as-

sociated with an increase in the urinary diastase according to Probstein and co-workers.[527]

A correct preoperative diagnosis can be made in a large percentage of cases. Correct preoperative diagnoses have been reported in incidences of 83.5 per cent,[330] 85 per cent,[591] 86 per cent,[551] 92.1 per cent,[355] and 95.3 per cent.[403] In the Charity Hospital series of 211 cases this incidence was 92.4 per cent. In a collected series of 2,322 cases[729] a correct preoperative diagnosis was reported in 1991 (85.7 per cent).

Probably the most common error in diagnosis is confusion with acute appendicitis. In Bager's[21] series of 1,495 cases 14 per cent and in Barbera's[29] series of 100 cases 11 per cent were diagnosed preoperatively as acute appendicitis. Eliason and Ebeling[179] reported a similar error in diagnosis in 10.8 per cent of 74 cases; Fallis,[192] Brown,[82] and McCreery,[403] in 7 per cent of 100 cases each; Jirásek and Persky,[312] in 20 per cent of 80 cases; Ross and Letourneau,[551] in 5.6 per cent of 228 cases; and Shawan,[591] in 1.8 per cent of 227 cases. Smith[611] in 1906 performed experiments in which he showed the direction of flow in the peritoneal cavity of intragastric contents following perforation. The passage of this material to the right iliac fossa and the consequent peritoneal irritation in this area probably account for the frequent clinical manifestations of appendicitis. In a collected series of 2,080 cases[730] acute appendicitis was the preoperative diagnosis in 141 (6.8 per cent) cases, acute cholecystitis in 47 (2.2 per cent), acute abdomen in 21 (1 per cent), intestinal obstruction in 17 (0.8 per cent), acute pancreatitis in 5 (0.2 per cent), pneumonia in 4 (0.19 per cent), coronary thrombosis in 2 (0.09 per cent), and a variety of other conditions in 73 (3.2 per cent). In the Charity Hospital series of 211 cases acute appendicitis was the preoperative diagnosis in 9 (4.3 per cent), acute cholecystitis in 2 (0.9 per cent), and in 1 each (0.47 per cent) the diagnosis was acute pancreatitis, acute intestinal obstruction, mesenteric thrombosis, and ruptured ectopic pregnancy. In addition to these conditions there are a number of others which should be considered in the differential diagnosis. However, this phase of the subject has been adequately discussed in numerous previous publications.

Baker[24] in 1920 and subsequently Nisbet[460] and Morrow[433] directed attention to the use of methylene blue as a means of facilitating recognition of the site of perforation at the time of operation. Accordingly 2 or 3 ounces of methylene blue are administered orally shortly before operation.

(To be concluded in the December issue. The references will accompany the last section.)

Review of Recent Meetings

REVIEW OF THE TWENTY-THIRD MEETING OF THE AMERICAN SOCIETY FOR THORACIC SURGERY, CLEVELAND, OHIO, JUNE 6, 7, AND 8, 1940

N. Logan Leven, M.D., St. Paul, Minn.

(From the Department of Surgery, University of Minnesota)

ON JUNE 6 the program was arranged as a joint session of the American Association for Thoracic Surgery and the National Tuberculosis Association.

Andre Cournand, D. W. Richards, Jr., and **Herbert C. Maier,** New York City: **Forms of Pulmonary Insufficiency Associated With Collapse Therapy.**—In patients with physical disability due to disease of the lungs or restriction of breathing apparatus, three general forms of insufficiency can be recognized: (1) failure of pulmonary ventilation, leading primarily to dyspnea (ventilatory insufficiency); (2) failure of respiratory gas exchange, leading to anoxemia and cyanosis (respiratory insufficiency); (3) combined failure of ventilorespiratory and of cardiocirculatory mechanisms, with variable symptomatology. Two cases are presented which illustrate different aspects of ventilatory, respiratory, and circulatory insufficiency associated with collapse therapy.

G. G. Ornstein, New York City, suggested a simple test for a surgical margin of safety; namely, if the maximum minute ventilation is six times the resting ventilation, surgery is safe. **Max Pinner,** Ithaca, N. Y., pointed out that the minute ventilation was important in reference to surgery depending on the distribution between both lungs. He stated that diaphragmatic paralysis produced the lowest breathing reserves, lowered the oxygen concentration, and increased latent pulmonary congestion; and that post-thoracoplasty scoliosis did diminish breathing reserves but did not produce respiratory insufficiency or cardiorespiratory impairment.

E. P. Eglee, R. H. Wylie (by invitation), and **Adrian V. S. Lambert,** New York City: **Empyema and Unexpanded Lung Problems in Pneumothorax.**—A report of a series of empyemas in the course of pneumothorax met with at Bellevue Hospital and in the Bellevue Clinic during the past five or ten years is presented. One hundred and thirty-six cases, or 7.8 per cent of all pneumothorax cases, developed empyema; the criterion for empyema being "thick pus" and not merely turbid fluid. The treatment consisted of drainage of the empyema by thoracotomy to reduce the surface of infection followed by thoracoplasty and later a Schede operation.

Louis Davidson, New York City, pointed out that two-thirds of these empyemas have a bronchopleural fistula and that this type of case when promptly treated as outlined offered 68 per cent cures. All thoracotomy patients not followed by thoracoplasty die. **Jerome R. Head,** Chicago, stated that most cases of empyema complicat-

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ing pneumothorax can be cured by conservative treatment if there is not too much contralateral disease. Daily irrigations with antiseptic solutions are used to sterilize the cavity, avoiding drainage and then performing a simple thoracoplasty. **A. V. Lambert,** New York City, pointed out that pneumothorax should not be taken lightly. Too often it meant giving a disease to the patient because of poor technique or too prolonged utilization of pneumothorax. He stated that cases of tuberculous empyema were treated too long medically before coming to surgery.

B. P. Potter, Jersey City, N. J.: **Hospital Mortality and Life Expectancy of Two Groups of Patients Treated With and Without Collapse Therapy.**—A comparison was made of two groups of patients: the first during the period between 1926 and 1932, when 6.2 per cent received collapse therapy; the second group treated in the interval between 1932 and 1938, when 61.2 per cent of the admissions were subjected to one or a combination of the procedures of pneumothorax, phrenic interruption, and thoracoplasty. The comparison of the total annual death rate and death rate per new admission in the two periods indicates a definite reduction in the total and per new admission death rate in the later years.

Jerome R. Head, Chicago, felt that the improvement since 1925 was not all due to collapse therapy but to some extent due to more strict rest regimen.

Minas Joannides, Chicago, stated that collapse therapy decreased the morbidity and also the risk of contact by augmenting the frequency of sputum conversion and causing the disappearance of cavities. He pointed out that the improvement of the patient and the death rate varied directly as did the time in the course of the disease at which the patient presented himself.

R. G. Bloch, W. B. Tucker (by invitation), and **W. E. Adams,** Chicago: **Standards and Criteria in Artificial Pneumothorax Therapy.**—This paper pointed out that in the management of the tuberculous patient some use pneumothorax universally; whereas, certain indications should be observed. Collapse therapy demands the presence of a cavity and should not be used in noncaseous, nonexcavated lesions. Of paramount importance is the combination of artificial pneumothorax with carefully supervised rest treatment. Sanitarium treatment and teaching are necessary for the patient. Ambulatory pneumothorax is not advised. The manner in which the overwhelming number of reports on results of artificial pneumothorax treatments have been made in the literature is largely responsible for an overenthusiasm as to its possibilities in curing tuberculosis. Few reports are based on the cognizance of the fact that the successful treatment means: (a) restoration of the lung to its physiologic function, i.e., complete re-expansion; (b) adequate roentgenologic evidence of healing of the tuberculous involvement, especially of the disappearance of cavities; (c) return of the patient to normal life, with (d) persistent absence of tubercle bacilli in the sputum; (e) persistent absence of all symptoms of activity; and (f) complete disappearance of all extrapulmonary complications.

Only after at least two years of satisfactory application of these criteria should a patient be considered as cured by the treatment. A distinct line is to be drawn between such final results and initial symptomatic improvement which is frequent and striking but also often misleading as to the ultimate outcome. The application of stricter standards in judging final results tends to reduce the percentage of "cures" considerably.

In the discussion **D. B. Cole,** Richmond, Va., stated that he found ambulatory pneumothorax satisfactory in many cases and that he thought it was important to get the patient back to work.

Oscar Auerbach and **Henry Green**, Staten Island, N. Y.: **The Pathology of Cavity Healing.**—This study based essentially on post-mortem material is qualitative and directed at the methods of cavity healing rather than quantitative in the sense of establishing a frequency of healing. Those cases in which the cavities undergo healing and death occurs as a result of the complications of chronic pulmonary tuberculosis as amyloid uremia, mixed infection empyema, or some cause other than tuberculosis have been included in this report. Cavities were regarded as closed clinically when the sputum became negative and serial x-rays failed to reveal a preexisting cavity. Healing of the tuberculous cavity occurred in two ways: (1) The lumen of the cavity was obliterated (''closed'' healing) and (2) the lumen of the cavity remained patent (''open'' healing). Closed healing results either in the replacement of the cavity by a scar or the retention or inspissation of caseated material within the cavity, thus filling and obliterating its lumen. The junction with the bronchus closes and the wall of the cavity closes concentrically with calcification. Healing with an open cavity lumen or open healing may occur in the presence of draining bronchi which open widely into the lumen of the cavity. Two factors occur in this form of healing: first, the complete shedding of the inner zone of caseation, resulting in disappearance of tubercle bacilli from the sputum; and, second, the transformation of the tuberculous granulation tissue of the cavity wall into a nonspecific fibrous tissue. These two processes probably go on simultaneously. Cavity drainage by means of the Monaldi operation may permit more rapid healing of the cavity.

Max Pinner, Ithaca, N. Y., states that these cases of closed healing did not actually represent healed cavities but were only ''cold abscesses.'' **H. C. Sweeney**, Chicago, stated that many people with tuberculous cavities that have healed died years later and the scar was overlooked.

Harold Brunn, Sidney Shipman, and **Alfred Goldman** (by invitation), San Francisco: **Intracavitation Studies in Tuberculosis.**—Pressure studies, examinations with the cavernoscope, and also bacteriologic studies of cavities as well as bronchoscopic examinations to determine the character of the bronchi were made. A method of study of cavities following somewhat that of Monaldi in Italy was presented. It was pointed out that the high tension in some cavities may account for some failures of collapse of cavities by thoracoplasty and decompression by needle or tube was suggested.

Leo Eloesser, San Francisco: **Further Experiences With Blocked Cavities in Pulmonary Tuberculosis.**—Manometric measurements by needling the cavity, the effect of pneumothorax on cavities, and the effect of inspiration and expiration and various postures on the shape and size of the cavity as seen in x-ray were studied. It was pointed out that collapse is theoretically wrong in blocked cavities and in these cases needling and aspiration of the cavity is advisable just prior to thoracoplasty.

Edward Kupka and **Edwin Bennett**, Olive View, Calif.: **Suction Aspiration of Tuberculous Cavities (the Monaldi Procedure).**—Closed cavity drainage has been limited to needling in connection with cavity research until its elevation by Monaldi into an important therapeutic measure. In 1938 he first employed continuous or intermittent suction over a prolonged period through a catheter draining the cavity. If disease changes in the parenchyma about the cavity have not progressed too far, the suction acts in several ways: (1) initially, drainage of cavity contents, such as pus, blood, detritus, and bacilli; (2) re-expansion of surrounding atelectasis; (3) reaeration of surrounding areas of exudative infiltration;

(4) compensatory expansion (emphysema) of normal pulmonary tissue within a contiguous area; (5) finally, holding of cavity walls in close apposition until symphysis takes place. An open or potentially open pneumothorax space is an absolute contraindication to cavity puncture because of the danger of empyema. To seal the pleural surfaces together, single or repeated injections are made of the patient's own blood into the pleural space, until repeated pneumothorax attempts over the area to be punctured with the trocar fail to elicit manometer fluctuations.

A tube is inserted and attached to intermittent suction with a trap for secretions. Treatment is continued until the cavity is closed and the catheter spontaneously extrudes with an upper limit of four to six months. Isolated balloon type cavities between 3 and 8 cm. in diameter lying in the subclavicular area with little surrounding parenchymal involvement offer the most favorable indication. In some instances closed cavity drainage may be used preparatory to thoracoplasty since cavities may reduce in size, sptutum become negative, and the general condition improve to a point where the major procedure can be done. A series of twenty-five cases was reported, the majority of which were still under suction.

In the discussion of this paper **G. G. Ornstein,** New York City, felt that the indications for the Monaldi operation were: (1) a giant ball-valve cavity; (2) bilateral disease; (3) marked decrease in pulmonary ventilation. **W. A. Hudson,** Detroit, stated that the Monaldi operation was of value in patients where thoracoplasty failed. It removed the secretion and prepared the patient for further surgery. In addition to drainage Hudson irrigates the cavity with a digestant aiding in the removal of slough and tuberculous granulation tissue.

David T. Smith, Durham, N. C.: **Treatment of Fungus Infections of the Lung.**—Many pathogenic fungi produce diseases in man which have a higher mortality than tuberculosis. In the treatment of fungus infection of the lung Smith indicated the value of iodides in actinomycosis, of gentian violet in pulmonary infection with monilia, of colloidal copper or antimony and potassium tartrate in cases of coccidioidal granuloma. He felt that sulfanilamide had proved to be of some value in the treatment of actinomycosis.

In discussion **O. H. Wangensteen,** Minneapolis, Minn., pointed out that actinomycosis might be endogenous or exogenous and that the actinomyces of the mouth resembled the others. Since the actinomyces is anaerobic, nonmotile, and carried by macrophages, he recommended simple curettement of the lesion, removal of the dead tissue, and exposure of the lesion to the air, indicating this as the major factor in the therapy of this disease.

Meetings on June 7 and 8 were then held in the auditorium of the Medical Library Association.

E. C. Janes and (by invitation) **D. B. Aitchison** and **A. Forsberg,** Hamilton, Ontario: **Extrapleural Pneumonolysis.**—This paper was based on the study of seventy-seven cases. There were eight deaths and in two-thirds of the cases there was closure of the cavity with sputum conversion. One-fourth of the cases had poor results and required later thoracoplasty. Fifteen cases were converted to oleothorax to maintain permanent collapse of the lung. Special reference was made to irrigation with Dakin solution of the extrapleural space prior to closing to avoid clotting with subsequent obliteration and secondary infection. This procedure is indicated where urgent collapse is necessary to control bleeding, where there is bilateral disease, and in young patients. It may also be useful to prepare a very ill patient for more drastic surgery. The presence of a large cavity close to the periphery, extrapulmonary disease, and low vital capacity are contraindications to this procedure.

John S. Harter and **Allen A. Lilienthal** (by invitation), Sanatorium, Miss.: **Extrapleural Pneumonolysis in Artificial Pneumothorax. A Report of 28 Cases.**—This series of cases included pneumothoraces which were not effective due to adhesions, the adhesions being of the type that could not be severed by the closed method or the open method of pneumonolysis. The parietal pleura was separated from the chest wall in the region of the adhesion and the pleura opened and cut around so as to leave parietal pleura attached to the free adhesions. The removal of the pleura around the adhesions transforms a small extrapleural pneumothorax into a complete intrapleural pneumothorax. In twenty-eight cases operated upon an effective pneumothorax was obtained in twenty-seven.

E. J. O'Brien, Detroit, felt that in most cases a thoracoplasty would be of more value than this type of procedure. **Minas Joannides,** Chicago, presented a motion picture of an axillary approach for this operation, pointing out that from the axillary approach one could work either anteriorly or posteriorly.

William A. Zavod (by invitation), New York City: **Bronchospirography. 1. A New Bronchospirographic Catheter and the Technique of Intubation.**—A double-channeled rubber catheter for intrabronchial intubation was described. The channel for the trachea was 9 cm. shorter than the channel for the bronchus and each channel had an inflatable bag which permitted separate respiration of each lung. Under local anesthesia the catheter was passed into the trachea and the left main bronchus. By inflation of the bronchial and tracheal bags studies could be made on each lung separately. This catheter is a modification of the Gebauer tube presented at a previous meeting.

George Leiner (by invitation), **Max Pinner,** Ithaca, N. Y., and **William Zavod** (by invitation), New York City: **Bronchospirography. 2. Application to Collapse Therapy.**—In a series of bronchospirographic observations made on patients with pulmonary tuberculosis it was found that neither clinical nor roentgenological findings are reliable indices of the functional capacity of the lung. It was found that collapse therapy might reduce the pulmonary ventilation without decreasing the oxygen consumption, indicating a better economy by compression of the diseased tissue only. Thus, there might be an increase of the ventilating efficiency in spite of a reduction in vital capacity. The authors felt vital capacities were entirely misleading and that bronchospirographic studies should be made in all cases before any irreversible collapse procedure was carried out.

S. O. Freedlander, Cleveland, stated that, in general, clinical and x-ray evidence parallels the bronchospirographic findings and that, while these findings might be decisive in an occasional case, the procedure would not become routine in estimating the surgical risk prior to collapse therapy.

David Salkin and **A. V. Cadden** (by invitation), Hopemont, W. Va.: **Rest and Collapse in Pulmonary Tuberculosis.**—This paper proposed a new classification of pulmonary tuberculosis. In using this proposed system a series of cases was studied to evaluate three different programs of therapy: (1) mild activity, (2) strict bed rest, (3) collapse therapy. The use of strict bed rest as a standard by which to measure other forms of treatment was suggested.

Emil Bogen (by invitation), Olive View, Calif., pointed out that collapse therapy produced a higher mortality than rest in minimal cases but that in advanced cases collapse therapy reduced the fatality rate.

A. V. S. Lambert, New York City: **Presidential Address. Etiology of Thin-Walled Cysts Occurring Within the Thorax.**—Intrathoracic cysts were divided

into three groups: parasitic, dermoid, and lymphangiomas. These lymphatic cysts probably arise as diverticuli from the pericardiocoelom by failure of the pericardial lacunae to fuse. They should be called pericardiocoelomic cysts. These cysts are usually discovered by x-ray and should be removed before pressure symptoms arise. They are thin-walled cysts lying anterior to the pericardium and lingula and may be shelled out clean by blunt dissection. They appear to lack any blood supply.

Peter Heinbecker, St. Louis: **Angina Pectoris.**—A general review of the mechanism of painful sensations and in particular those arising in the heart was presented. Whereas pain endings in the skin are easily excited by any stimuli, those in the viscera are excited by tension and tissue metabolites. Ischemia of cardiac muscles due to occlusive arterial disease will produce metabolites which stimulate pain endings. Muscular effort and anoxemia increase production of metabolites and similarly produce stimuli. The pathways of the pain were traced. This cardiac pain is helped by the usual rest, vasodilator drugs, and morphine. Although total ablation of the thyroid reduces the work of the heart, it also tends to increase arterial degeneration and three-fourths of these cases die of rupture of the aorta. Alcohol injections of the ganglia or excision of the inferior cervical and first and second thoracic ganglia offer some relief of the pain. Establishment of collateral circulation to the heart by muscle or omental transplants to the heart or by the establishment of an adhesive pericarditis has shown encouraging results.

Claude S. Beck, Cleveland, Ohio, pointed out that neither metabolites nor anoxemia alone would produce cardiac pain, but together they would increase the pain, one augmenting the other. While general anoxemia was well tolerated, local anoxemia was devastating to the heart. Experimentally, he found that simultaneous cervical sympathectomy with ligation of the left coronary artery reduced the mortality from 80 to 50 per cent, but that cervical sympathectomy sixteen days after ligation of the arteries offered no protection.

Arthur S. W. Touroff, New York City: **The Surgical Treatment of Patent Ductus Arteriosus Complicated by Subacute Bacterial Endocarditis.**—Subacute bacterial endocarditis is readily superimposed in cases of patent ductus arteriosus. The infection is chiefly in the left side of the heart on account of the higher oxygen content which allows better growth of the *Streptococcus viridans.* Thus, excision of the patent ductus may remove a focus of infection and decrease the infection in the pulmonary artery by reducing the oxygen tension. Operation in these cases is complicated because of the fact that the periarteritis usually makes the ductus adherent to the pulmonary artery. This, together with degenerative atheromatous plaques, makes the ductus friable. Four cases were reported; one patient has been free from all evidence of the original lesions for more than three months; two died of hemorrhage and the fourth was improved.

Frank S. Dolley, Los Angeles, indicated that the risk of postponing operation in cases of patent ductus arteriosus was greater than the risk of operation due to the probability of the development of subacute bacterial endocarditis. However, he felt that operation in cases of subacute bacterial endocarditis was of doubtful value since, as a rule, the infection had extended into the conus. He advocated early operation before vegetations formed and presented nine cases of children who were well following ligation of a patent ductus arteriosus. In none of these was infection present at the time of operation. **J. W. Strieder,** Boston, said that chemo-

therapy did no good in bacterial endocarditis and that no cures have been reported since 1924. In these otherwise hopeless cases this operation should be tried.

Frederick R. Mautz, Cleveland, Ohio: **Mechanism for Intermittent Pulmonary Insufflation in Thoracic Surgery.**—A respirator built into a gas machine for intermittent pulmonary ventilation was demonstrated. Intermittent pressure is more efficient in open pneumothorax than the continuous flow mechanism. This machine gives perfect control of an open pneumothorax because it is visibly adjustable.

Claude S. Beck, Cleveland, Ohio: **Exhibit of Experimental Work on the Heart.**—A specimen was demonstrated by slides to show definite collateral circulation of the coronary with the internal mammary arteries, after ligation of the coronary arteries and transplantation of a muscle fat graft in the experimental animal.

Harold Feil, Cleveland, presented a follow-up study of 30 cases of coronary sclerosis operated upon by Beck, following these over a period of from five years to three months after operation. In this series there were twenty-nine men and one woman of an average age of 51 years. All of these patients had failed to respond to medical treatment, which consisted chiefly of administration of nitroglycerin and aminophyllin. While there was no immediate operative mortality, ten patients died in the period a few hours to a few months following the operation, giving a 33 per cent mortality. Of the patients surviving operation an excellent result was obtained in ten; that is, the patients were able to return to work; in seven the result was considered fair, the patients having moderate pain relief, and in three there was no relief of pain. There may be relief of pain and the weakness may continue. **J. W. Strieder,** Boston, presented a case of coronary sclerosis that was improved six months following an omental graft after the method of O'Shaughnessy. Gastric symptoms developed at that time due to a hiatus hernia of the stomach through the site of the transplant of the omentum. The patient died forty-five minutes after the reduction of this hernia at operation. A rich vascular supply was found present between the omentum and the heart.

A. L. Brown (by invitation), San Francisco, demonstrated a modification of the Jackson bronchoscope. Light was transmitted to the end of the bronchoscope through a lucite carrier. By this method a slightly larger working space was obtained in larger tubes. The possibility of diversion of the light in any direction from the end of the lucite might prove to be of some advantage.

W. H. Stewart, H. C. Maier, C. W. Breimer (by invitation), New York City: **Ciné-Roentgenographic Studies of the Pulmonary Circulation, the Chambers of the Heart, and the Greater Blood Vessels, in Health and Disease.**—By the rapid injection of diodrast into the veins, very interesting motion pictures were made of the pulmonary circulation, chambers of the heart, and greater blood vessels. The possibility of use with this method in diagnosis was pointed out.

Brian Blades, St. Louis: **Individual Ligation Technique for Lower Lobe Lobectomy.**—A technique involving separate ligation of each anatomical structure in the hilum of the lung is recommended for lower lobe lobectomy. The method is based on investigations of the surgical anatomy of the lower lobes and experiences with eleven successful cases in which the procedure was employed. Using this method a higher incidence of permanent bronchial closure was obtained and a decrease in the number of empyemas was found. In eleven cases five did not have empyema and six had empyema but not the putrid type. Five of these had

a small bronchial fistula as demonstrated by saline injection. However, this did not prolong the hospital stay. In these cases an average hospital stay was thirty days. By the individual ligation technique the danger of compression or encroachment on the middle and upper lobe bronchi that occasionally obtains when mass ligation of the lobe or hilum is carried out may be avoided.

Harold Brunn, San Francisco, pointed out that mass ligation of the lobe or hilum was not clean surgery and that, if the condition of the patient permitted, the individual ligation technique should be used. He felt that formation of adhesions to the upper lobe as a preliminary procedure to the operation made the postoperative course easier. **R. M. Janes,** Toronto, Ontario, pointed out that failure of the middle and upper lobes to expand after lower lobe lobectomy may be accounted for by encroachment on these bronchi occurring when mass ligation of the hilum is used.

J. J. Singer, John C. Jones, and **L. J. Tragerman** (by invitation), Los Angeles: **Aseptic Pleural Adhesions Experimentally Produced.**—In this study by injecting various substances in the pleural spaces of rabbits aseptic adhesions were successfully produced. The best results were obtained using iodized talc in saline solution. By this method the saline was readily absorbed and left an even deposit of talc. The adhesions produced were particularly effective in the fixation of the mediastinum which represents the most vulnerable area of infection following lobectomy. It was pointed out that the risk of thoracic surgery is reduced by the preliminary treatment of the pleura by this method.

Clarence Bird, Louisville, Ky., injecting silicon into the pleural cavity, found that more effective adhesions were formed if a phrenicectomy had been performed and if the pleural cavity was kept dry. **Cameron Haight,** Ann Arbor, Mich., used iodized talc to make adhesions prior to lobectomy. He advised using a minimum amount of the powder, just sufficient to produce a sheen over the surface. If too much powder is used, it collects in masses. **L. J. Tragerman,** Los Angeles, indicated that the adhesions probably were not as important as the inflammatory blocking of the lymphatics.

Frank S. Dolley and **John Jones,** Los Angeles: **Experiences With Lobectomy and Pneumonectomy in Pulmonary Tuberculosis.**—In this paper seven cases were reported, four total pneumonectomies and three lobectomies. Of the four cases of pneumonectomy two are dead and two are dying with contralateral spread, while the three cases of lobectomy are still alive and well. Preliminary thoracoplasty with apicolysis was advised to improve the preoperative condition. Thoracoplasty also obviates the need for obliteration of the dead space following pneumonectomy or lobectomy. Individual ligation of the hilus, catheter suction of the trachea, and minimal handling of the lung were advised. If the lung is adherent, ligation of the hilus should be done as the first step to prevent contralateral spread by inoculation of the infection. It is important to make the patient cough postoperatively to get rid of secretions.

John Alexander, Ann Arbor, Mich., reported cases of bronchostenosis treated by thoracoplasty with good results. He pointed out that in some cases the stenosis might be so small that a preliminary thoracoplasty prior to lobectomy would be dangerous. If ulcerative tracheobronchitis due to tuberculosis cannot be entirely removed by this operation, it is contraindicated. He reported one pneumonectomy and two lobectomies for tuberculosis which apparently are cured.

Evarts Graham, St. Louis, pointed out that, if the chest wall is not collapsed by thoracoplasty, a load is put on the opposite lung by a lobectomy.

Leo Eloesser, San Francisco, found thoracoplasty disappointing in cases of bronchostenosis and pulmonary suppuration and preferred either drainage or lobectomy in these cases. He advised lower lobe lobectomy for bronchiectasis that developed after upper lobe thoracoplasty and indicated that most of these cases of bronchiectasis were probably not due to tuberculosis.

J. J. Longacre and **Ralph Johanson** (by invitation), Cincinnati, Ohio: **An Experimental Study of the Fate of the Remaining Lung Following Total Pneumonectomy.**—Hyperplastic regeneration of the remaining lung following total removal of one lung occurs in young animals in contrast to the dilatation of the definite lobules occurring in adult animals. Following pneumonectomy, the cardiorespiratory reserve is cut in half, sufficient for rest and moderate exercise but inadequate when these animals are subjected to strain. In nine to twelve months the cardiorespiratory reserve returns to 70 or 80 per cent of normal in the adult animals; whereas, in animals pneumonectomized as puppies the return is 100 per cent, showing that capacity of the organism for further growth accounts for the difference in the anatomical changes and also the difference in function. In animals pneumonectomized as adults there was noted dilatation of the atria and the alveoli at the periphery of the lung with some breaking of alveoli and elastic fibers causing emphysema. It was pointed out that thoracoplasty might prevent this dilatation in human beings subjected to pneumonectomies.

Evarts Graham, St. Louis, indicated that thoracoplasty in cases of pneumonectomy would prevent kinking of the great vessels and the trachea to relieve discomfort and dyspnea. **W. E. Adams**, Chicago, studied the effect of paralysis of the diaphragm and thoracoplasty in animals having lobectomies and pneumonectomies. He found that there was less dyspnea in cases with section of the phrenic nerve. In one animal with both diaphragms paralyzed and bilateral lung resection, leaving but one lobe, he found a normal reaction to conditions simulating an ascent to 27,000 feet altitude. The animal did not lose consciousness.

Chevalier L. Jackson and **A. R. Judd** (by invitation), Philadelphia: **The Role of Bronchoscopy in the Treatment of Pulmonary Abscess.**—The causes of lung abscess and the bacteriology of these abscesses were reviewed in a series of 137 cases. The role of bronchoscopy during several stages of lung abscess was indicated. First, as a prophylactic measure lung abscess may be prevented by aspiration of material from the bronchus. Second, as a diagnostic measure bronchoscopy may aid in the differentiation of a tuberculous cavity by obtaining material for bacteriologic study and also may identify a tumor in the bronchus as an etiologic agent. Third, during the acute stage bronchoscopic drainage may lead to spontaneous healing. It is emphasized, however, that bronchoscopic and so-called nonsurgical treatment should not be carried beyond the optimum time for the employment of some other procedure such as surgery. **P. C. Samson**, Oakland, Calif., stated that next to cancer lung abscess was the most fatal common disease in the chest. He advised bronchoscopic examination of all cases except those complicated by hemorrhage or brain abscess. If improvement does not occur within a few days, surgical treatment should be used. The more chronic and the more putrid the abscess, the less likely it is to improve by bronchoscopic drainage.

Paul W. Gebauer, Cleveland, Ohio: **The Differentiation of Bronchiogenic Carcinomas.**—Pathologically and clinically small-cell carcinoma, adenocarcinoma and squamous-cell carcinoma are the three fundamental types of bronchogenic

cancer. The differences of these types early in the course of the disease sometimes permit their distinction by combined clinical, radiologic, and bronchoscopic investigations. Late in the disease this is true in approximately 60 per cent of cases. Bronchoscopy will be negative in 40 or 50 per cent of cases if performed at the onset of symptoms. When negative, it should be supplemented by other biologic, endoscopic, and radiographic diagnostic methods until the cause for symptoms is known. The impression that clinical symptoms tend to occur early, when the tumor is in an operable state, has been gained from this study. Therefore, it is felt that if the diagnosis is made early and there is the proper selection of operative material, surgical treatment will gain a respectable position in therapeusis.

Evarts Graham, St. Louis, in discussion, stated that squamous-cell carcinomas of the bronchi are a definite group, but it is impossible to differentiate the small-cell carcinomas and adenocarcinomas, since these varied depending on the portion of the tumor examined. Small-cell carcinomas, adenocarcinomas, and the adenomas probably arise from remnants of fetal bronchial buds. The adenomas, he said, were potentially malignant and should not be removed locally. **Alfred Goldman,** San Francisco, found one case in fifty adenomas that metastasized; fifteen cases having a fifteen-year history showed no evidence of metastasis, the growth being by local extension.

Henry K. Beecher (by invitation), Boston: **Controversial Points in Anesthesia for Thoracic Surgery.**—Of the anesthetic agents, Beecher advised using ether since it could be used with 96 per cent oxygen; whereas, cyclopropane was used with 75 per cent oxygen. Cyclopropane may be toxic to cells, interfering with oxygen use. Toxic cardiac effects are noticed after the use of cyclopropane for one hour; the pulse rate increases and the blood pressure drops. Ether is well tolerated with circulatory impairment and that it is irritating is of no importance since it does not affect the mortality or morbidity in his experience. The objections to spinal anesthesia were the (1) inability to aspirate secretions from the trachea, (2) inability to give positive pressure effectively, (3) tendency to suppress cough, and (4) greater mortality with its use. In many cases an intratracheal tube is important because it offers an open airway for positive pressure and for aspiration of sputum. He felt that it was contraindicated in cases of pulmonary tuberculosis. Endobronchial catheters in anesthesia offer technical difficulties and may come out at inopportune times. The inflatable cuff on intratracheal catheters was criticized as unnecessary and in the presence of a gush of pus or blood might cause spillage of contents into the other side, which otherwise would come up alongside the tube. Mechanisms for controlled respirations were considered inadvisable and unnecessary.

H. R. Decker, Pittsburgh, stressed the use of local anesthesia supplemented by cyclopropane or by sodium pentothal. He avoided the use of ether in cases of pulmonary tuberculosis. **G. E. Lindskog,** New Haven, Conn., favored the use of avertin and intratracheal cyclopropane, using nitrous oxide anesthesia if local anesthesia alone was inadequate when cautery was to be used. He pointed out that ether was explosive if there was a high oxygen content in the mixture.

Howard K. Gray and **I. C. Skinner** (by invitation), Rochester, Minn.: **The Operative Treatment of Cardiospasm.**—In over 1,200 cases of cardiospasm seen at the Mayo Clinic, operations for the relief of cardiospasm were performed only seven times. These cases were reported. Mikulicz' operation of manual dilatation of the cardia was performed four times, esophagogastrostomy one time, thoracocervical sympathectomy one time, and abdominal sympathectomy com-

bined with manual dilatation of the cardia one time. In cases in which the esophagus is markedly dilated, lengthened, and tortuous (sigmoid esophagus) with the development of a reservoir below the opening of the cardia, surgery at times will be found necessary. The technique of abdominal esophagogastrostomy was illustrated and the case reported.

H. J. Moersch, Rochester, Minn.: **The Injection Treatment of Esophageal Varices.**—A case of esophageal varices successfully injected with a sclerosing solution through an esophagoscope was reported. A review of the various treatments used for esophageal varices was presented. Since fatal gastrointestinal hemorrhage occurs in 80 per cent of all esophageal varices and 50 per cent of all cases of cirrhosis of the liver die of hemorrhage, some method of therapy in these cases is highly desirable.

Alton Ochsner and (by invitation) **Michael Debakey** and **Samuel Murray,** New Orleans, La.: **Surgical Aspects of Carcinoma of the Esophagus.**—Three cases of carcinoma of the esophagus operated upon by the authors were reported. One patient with carcinoma of the esophagus in whom an excision of the lesion and an esophagogastrostomy was performed lived over one year with no evidence of recurrence. A transthoracic approach was used removing the left eighth rib, crushing the phrenic nerve and opening the diaphragm. A wide resection of the stomach was carried out. The most desirable procedures for lesions at various levels in the esophagus were considered and illustrated.

B. Noland Carter and (by invitation) **Jean Stevenson** and **Osler Abbott,** Cincinnati, Ohio: **Esophagogastrostomy. An Experimental Study.**—Two successful cases of esophagogastrostomy were reported. Clinically the chief danger of esophagogastrostomy is leakage at the line of suture and this is most commonly due to tension. A method to obviate tension on the anastomosis has been devised. This consists of anchoring both the stomach and the esophagus to the periosteum of nearby ribs and vertebral bodies. A second complication of stricture at the site of an anastomosis may be avoided by making the anastomosis to a circular hole in the stomach. This was indicated by experimental work.

Book Reviews

A Manual of Otology, Rhinology, and Laryngology. By Howard C. Ballenger, Assistant Professor of Otolaryngology, Northwestern University School of Medicine. Pp. 302, with 90 illustrations and 4 color plates. Philadelphia, 1940, Lea and Febiger. $3.75.

This manual is intended for the undergraduate medical student. It is a condensed version of the well-known Ballenger *Textbook on Diseases of the Nose, Throat and Ear*, the seventh edition of which appeared in 1938. Dr. Howard Ballenger was the junior author of that text which was written originally by Dr. William L. Ballenger.

The manual is a little less than one-third the length of the textbook. Its purpose is to emphasize anatomy, etiology, symptoms, and diagnosis. Surgical technique is omitted. A chapter on bronchoscopy which appeared in the textbook is entirely omitted.

The subject material is dealt with in four parts. The nose and accessory sinuses are treated in eleven chapters which are covered in 80 pages; the pharynx and fauces are covered in five chapters totaling 134 pages; diseases of the larynx receive six chapters of 34 pages; and the ear is treated in thirteen chapters of 121 pages.

There have been few manuals of this type available to undergraduate students. Consequently, this edition helps to supply a need. It is well written and illustrated and emphasizes the essentials which should be the basic part of undergraduate instruction.

Orthopedic Operations (Indications, Technique and End Results). By A. Steindler. Pp. 766, with 322 illustrations. Springfield, Ill., 1940, Charles C. Thomas, Publisher. $9.

The book is divided into three portions. The first, on general biological and clinical topics pertaining to orthopedic operations, comprises 66 pages and includes 32 pages devoted to operative approaches to bones and joints. The second section of 305 pages is devoted to descriptions of the technique of various orthopedic operations and comprises twelve chapters. It includes surgery of tendons, fascial and ligamentous surgery, surgery of bones, joints and nerves, skin, and the spine. Amputations are included in the section on the surgery of bones.

The third section is devoted to the indications for specific operations and end results in various orthopedic conditions. This includes nine chapters which are devoted to congenital deformities, traumatic and constitutional deformities, inflammatory conditions, chronic arthritis, disturbances of the nervous system, degenerative bone diseases, tumors, and circulatory diseases.

The book is based on the experience of the author's clinic at the University of Iowa and in both the second and third portions statistics are given as to the results obtained in this clinic from many operative procedures. These results are classed as good, fair, and poor. In the third section similar statistics are given as to the results obtained by the operative treatment of specific conditions. These statistics represent a vast amount of looking up of histories and this work appears to have been done by the author's students and assistants. However, it is the reviewer's opinion that such bare statistics are of relatively little value.

In Section 3 reference is made to the operative procedures in Section 2. Unfortunately, in these references to the operative procedure the page is not given, but one is referred back to Part 2, Chapter X, etc., as the case might be, and it is troublesome to look up the operative technique. Likewise, in the description of the operative technique in Part 2 one is referred back to Part 1 for a description of the operative incision.

It is thus evident that this is not an extensive operative surgery, because, in attempting to cover both the operative technique and the indications for specific operations in the various orthopedic conditions, the author has devoted more than one-half of the book to the latter and has limited the part of the book on operative technique to approximately 300 pages. Naturally, many operations are omitted. In the preface the author states that this is not because these operations were not known to him, but because their validity did not impress itself sufficiently upon him. Most orthopedic surgeons will differ with any other orthopedic surgeon as to what operations are worthy of inclusion in a book, the title of which is *Orthopedic Operations*, because they all have their preferences.

The book is rather sparsely illustrated, there being 322 illustrations, of which only 100 are included in Part 2, which is devoted to operative technique. The illustrations in Part 2 are mostly original drawings and are clear and illustrate the point which they are intended to convey, although, for the most part, they are rather sketchy as to the anatomy of the region. The same is true of the description of the various operations. It is believed that in a book of this sort the technique of the various procedures should be described in detail and that the difficulties which may be encountered should be mentioned so that a competent surgeon may be in a position to perform a given operation after he has read its description. However, if the surgeon has the time and access to a good medical library he can look up the detailed description of the various procedures which are mentioned, because the value of the book is enhanced by a list of references which is placed at the end of each chapter.

On the whole this is a very valuable and full record of Dr. Steindler's work at Iowa City. For this reason it will be welcomed by American orthopedic surgeons.

Chronic Diseases of the Abdomen. A Diagnostic System. By C. Jennings Marshall. Cloth. Pp. 247, with 131 illustrations. Boston, 1939, Little Brown & Company. $6.

This book of 247 pages presents a plan of study of the chronic diseases which occur within the abdomen, with a consideration of the symptomatology of diseases outside of the abdomen which must be differentiated from abdominal disease. The work is divided into two parts. Part I covers 48 pages and is devoted to methods of diagnosis. Differential diagnosis is considered in Part II, which fills the remainder of the book. The value of the book lies chiefly in the second part, although some important diagnostic methods are described in the first part. The differential diagnosis of chronic abdominal disease is approached with pain as the guiding symptom. At first the diseases which may cause general, vague, and midabdominal pain are considered. Following this the possibilities of disease in all sections of the abdomen and back are considered when pain is a symptom in each locality. All of these are thoroughly discussed.

At the end of the book several pages are devoted to signs and symptoms which may or may not be associated with pain. These are hematemesis, ascites, variations in appetite, loss of weight, jaundice, chronic diarrhea, hematuria, vomiting, pyrexia in abdominal disease, and abdominal tumor.

A differential diagnosis of any diseased condition cannot be made unless the possibilities are considered. In this book the possibilities are mentioned and are briefly or fully considered as the importance of the location of pain in the abdomen seems to warrant. This is a valuable book, logically arranged for teaching and a ready reference in the study of chronic abdominal disease.

Surgery of the Alimentary Tract. By Sir Hugh Devine. Cloth. Pp. 1,046, with 690 illustrations. Baltimore, 1940, Williams and Wilkins Co. $15.

In this volume of more than 1,000 pages the author has not attempted to develop systematically the surgery of all the contents of the peritoneal cavity. His principal concerns have been the surgery of ulcers and of malignancies of the colon and rectum. He does describe, however, operative procedures upon the biliary tract, spleen, and other accessories to the alimentary tract proper.

The work is written in four parts. The basis of this division is by no means clear. Subdivision of the subject matter on a more consistent plan or along more orthodox lines would have made the book far more readable.

The special value of the work lies in the description of technical procedures to which the author has given obviously much time and thought. All surgeons having a keen interest in gastric or colonic surgery will find much of value in these pages, which are in essence a digest of Devine's experience. They will be somewhat disturbed to note that an analysis of the author's results, even in the chapters of his own special interests, is not contained in the volume.

The book can be recommended enthusiastically, not as a source book, nor even as an authoritative work of reference on surgery of the alimentary tract, but rather as the résumé of the experience of a surgeon, whose ideas and technical contributions to certain aspects of surgery of the alimentary tract are of the greatest interest.

An Introduction to Gastro-Enterology. By Walter C. Alvarez, M.D. Ed. 3. Cloth. Pp. 778, with 186 illustrations. New York, 1940, Paul B. Hoeber, Inc. $10.

This book, published previously but under the title *Mechanics of the Digestive Tract*, appears now in enlarged form under the modest title of *An Introduction to Gastro-Enterology*. As the preface of the author indicates, the monograph is intended as a source book for various chapters of clinical physiology of the gastrointestinal canal. The admirable task which the author has set himself he discharges faithfully. Like earlier editions, it deals principally with the problems of motility of the gastrointestinal canal. It is in no sense a complete treatise on any of the common clinical disorders of the digestive tract. Yet, the writer, because of his experience as both investigator and clinician in the province of gastroenterology, has compounded a book that will be extremely useful for purposes of orientation to both physiologists and clinicians. The book concludes with an informative chapter on books of reference for various aspects of gastroenterology which the reader will welcome. A long and carefully selected bibliography is appended. The book can be recommended enthusiastically for the purpose for which its author intended it; namely, as an orientation source book for many chapters in physiological aspects of gastroenterology.

The Surgery of Injury and Plastic Repair Work. By Samuel Fomon, M.D. Cloth. Pp. 1,409, with 925 illustrations. Baltimore, 1939, Williams and Wilkins Co. $15.

This is in many respects a remarkable book. The first 500 pages are devoted to the general considerations of operative technique, tissue transplantation, wounds, burns, fluid and electrolyte requirements of the surgical patient, shock, anesthesia and pre- and postoperative management. The remaining 900 pages deal with special chapters of what is commonly described as plastic surgery.

The general surgical consideration which precedes the accounts peculiar to reconstructive surgery in the various regions is particularly good and complete. This reviewer feels constrained to say it is the best account of surgical principles that he has found in a treatise dealing with a special province of surgery. If this book is a reflection of the plastic surgeon's interest in the general principles of surgery, one may ask with justification, is the plastic surgeon to become the general surgeon of tomorrow, entrusted with the responsibility of carrying on the traditions and teachings of general surgery?

The historical accounts in many of the chapters are unusually good and complete. This reviewer would have anticipated finding in a book of this standard of excellence a more complete discussion of the microscopic aspects of wound healing, including consideration of ligatures and strengths of the coaptated tissues at various phases in the healing period. The plastic procedures of genitourinary, gynecologic, intestinal, and orthopedic surgery are not described. A valid criticism which with all fairness may be leveled against the text is lack of uniformity in illustrations. Though the text is illustrated generously, in the main, the illustrations are not of the quality which one would anticipate finding in a text of plastic surgery.

A well-selected bibliography accompanies each chapter and a useful bibliography completes the text. The book may be recommended as a useful source book to both elementary and advanced students of surgery.

Fractures and Other Bone and Joint Injuries. By R. Watson-Jones, B.Sc., M.Ch., Orth., F.R.C.S. Cloth. Pp. 723, with 1,040 illustrations. Baltimore, 1940. Williams & Wilkins Co. $13.50.

This book of 723 pages covers in a comprehensive and unusually clean-cut way the subject of injuries to the bones and joints. It is divided into six parts. In Part I the repair of fractures, delayed and nonunion, adhesions and joint stiffness, soft tissue, vascular and nerve injuries complicating fractures, clinical and radiologic diagnosis, the reduction and immobilization of fractures, and open and infected fractures are discussed. This section is perhaps the most outstanding in the book and abounds in facts and information of fundamental importance. Pathologic and birth fractures comprise Part II. Injuries of the head and trunk, with special emphasis on fractures of the spine and head, are discussed in Part III. This section of the book is also outstanding by reason of the lucid manner in which the material it contains is presented. Part IV takes up injuries of the upper limb, and Part V, injuries of the lower limb. While the material in these sections is excellently presented, they contain little that is new; unfortunately, internal fixation as a method of treatment in fractures of the extremities is largely ignored. The discussion of new and unusual cases in Part VI is interesting but does not add greatly to the value of the book. The appendix, which offers a plan of organization of a fracture service, contains some very valuable suggestions

and is worthy of careful study by those interested in organized fracture treatment. The index of authors and a general index provide a comprehensive bibliography of present-day fracture treatment.

Throughout the book, adequate immobilization of fractures, the necessity of avoiding unnecessarily immobilizing adjacent joints, the importance of active use in restoring function, and the ineffectiveness and harm of passive motion are constantly emphasized. The soundness of the views expressed cannot be questioned, and the precepts laid down are worthy of wide dissemination among those interested in fracture treatment.

The subject matter throughout is presented in a clear, concise manner and leaves no doubt in the mind of the reader as to what the author intended to express. This is true to an unusual degree. The illustrations and the x-ray reproductions are excellent and numerous and illustrate the text splendidly.

As a treatise on the nonoperative management of fractures and joint injuries, this book is important in a day when nearly every book on fracture treatment deals so largely with open reduction and the use of internal fixation. It is for this reason as well as the soundness of the principles it emphasizes that this book can be highly recommended as an excellent addition to the library of anyone interested in fracture treatment.

Die Chirurgie des praktischen Arztes unter besonderer Berücksichtigung der kleinen Chirurgie und der dringlichen Chirurgie. By Erich Sonntag. Ed. 2. Pp. 1,016, with 780 illustrations. Leipzig, 1939, Georg Thieme. 46 marks.

This is a textbook of general surgery written primarily for the students and practicing physicians of Germany. The book is divided in two parts, the first of which, comprising almost one-half of the text, is concerned with surgical principles and fundamentals, such as asepsis, anesthesia, hemorrhage, infections, and wound healing. The second half is devoted to surgical affections of various organs and is arranged in classical regional style beginning with the head and ending with the feet. About thirty pages at the end of the book are devoted to various drugs used in surgery.

The minor surgical procedures are well presented, but the author has made little attempt to include many recent advances in major surgical affections. Thus, for example, decompression by suction applied to an inlying duodenal tube is not even mentioned in the treatment of intestinal obstruction. In the discussion of parasitic diseases the author's statement regarding the transmission of *Taenia solium* is not in accordance with accepted facts. In the chapter on diseases of the liver the important subject of amoebic abscess is merely mentioned. However, the book may be readily recommended to the student as a manual of minor surgery. It is concisely written and the numerous illustrations are well reproduced.

Synopsis of Obstetrics. By Jennings C. Litzenberg, M.D., F.A.C.S. Pp. 376, with 157 illustrations, with 5 in color. St. Louis, 1940, The C. V. Mosby Co. $4.50.

This excellent little volume of 376 pages and 157 illustrations, including 5 in color, is heartily recommended as an introduction to obstetrics.

The organization of the material is extremely good and is presented in a very clear, concise, and intelligent manner. Despite the fact that the volume is a synopsis, a large amount of valuable material is included which cannot be found in larger and more voluminous textbooks.

Throughout this volume, the author's well-founded conservatism is reflected.

The illustrations are good and materially enhance the value of this volume.

Frank Howard Lahey Birthday Volume, June 1, 1940. By many authors. Cloth. Pp. 466, with 68 illustrations. Springfield, Ill., 1940. Charles C. Thomas, Publisher.

On the anniversary of his sixtieth birthday, a number of friends and colleagues contributed this collection of essays, about fifty in number, to do honor to one of America's versatile surgeons who has become widely known. The essays embrace a wide variety of subjects. The majority of the papers, however, concern well-known interests of Dr. Lahey; namely, surgical aspects of diseases of the stomach and duodenum, colon, biliary tract, and the thyroid gland.

Two of the papers are of unusual interest. One of these, under the authorship of Aaron and Murphy, of Buffalo, describes the gastroscopic reappearance of a normal gastric mucosa in patients having pernicious anemia and an atrophic gastric mucosa, following treatment with liver extract. The other, a paper written by Ivy, of Chicago, under the title of "Gastro-Intestinal Hormones," describes very briefly a new hormone which Ivy designates as urogastrone, a substance recovered from male and female urine, which on intravenous injection inhibits the gastric secretory response to histamine.

Ten papers in the volume concern themselves with varying aspects of the ulcer problem. The divergence of views expressed in these papers is striking and suggests how unsettled the problem is.

A Textbook of Bacteriology. By Hans Zinsser, M.D., and Stanhope Bayne-Jones, M.D. Ed. 8. Cloth. Pp. 990, with 115 illustrations. New York, 1939, D. Appleton-Century Co. $8.

Since its first appearance in 1910, this book has gone through eight editions. New developments in bacteriology and immunity have necessitated the inclusion of much new material and the revision of old. In many respects, therefore, this eighth edition is a new book as compared with the seventh edition of 1934.

Intended primarily for undergraduate students of medicine, the book at the same time should have a wide appeal to practitioners of medicine. Much of the subject matter is taken up with the morphology of bacteria, classification, the properties of bacteria and the effect of physical and chemical agents on bacteria. There is much material of a decidedly practical nature in the book, of particular interest to those whose function it is to apply in practice the principles of bacteriology and immunity. The introductory chapter on the history of bacteriology is especially deserving of mention. The book is well written and the subject matter has been subdivided carefully on a well-ordered plan. The collaboration of the two well-known Professors of Bacteriology, Zinsser and Bayne-Jones from the medical schools of Harvard and Yale Universities, has produced an excellent and authoritative text.

Treatment of War Wounds and Fractures, With Special Reference to the Closed Method as Used in the War in Spain. By J. Trueta, M.D. Foreword by H. Winnett Orr, M.D. Cloth. Pp. 148, with 48 illustrations. New York, 1940, Paul B. Hoeber, Inc. $2.50.

This small monograph presents a summary of the author's experience in the treatment of war wounds in the recent Civil War in Spain. The book contains an excellent historical account of the development of the treatment of open wounds of the extremities by immobilization in casts of plaster of Paris. Special con-

sideration is given the technical details of the management of injuries involving the various bones and joints of each extremity. The book can be recommended as an important contribution to the importance of immobilization in the management of injuries of the extremities.

Artificial Pneumothorax: Its Practical Application in the Treatment of Pulmonary Tuberculosis. Edited by Edward N. Packard, M.D., John N. Hayes, M.D., and Sidney F. Blanchet, M.D. Foreword by E. R. Baldwin, M.D. Cloth. Pp. 300, with 85 illustrations. Philadelphia, 1940, Lea and Febiger. $4.

Seventeen Saranac Lake physicians have collaborated to produce this 300 page monograph. It is essentially a recital of the personal experience and opinions of the authors as to the value of artificial pneumothorax in the treatment of pulmonary tuberculosis. This in itself is enough to commend the book to all who are interested in the treatment of tuberculosis. The text has been amplified with 85 illustrations and 33 statistical tables. Many of the photographs are of x-ray films and are unusually good. The present-day status of pneumothorax treatment in all its phases has been creditably summarized. There is nothing new. Controversial points are presented from an objective viewpoint. Extensive bibliographies are found at the end of each chapter which enhance the value of the book. Chapters on the selection of cases and the complications of pneumothorax will prove of considerable help and guidance for the physician who treats patients with pulmonary tuberculosis.

Biochemistry of Disease. By Meyer Bodansky, M.D., and Oscar Bodansky, M.D. New York, 1940, The Macmillan Co.

This work is of particular interest for the clinician, because biochemistry, although considered in every detail, is taken up only from the standpoint of clinical application. Frequently a clinician is lost in the maze of biochemistry, but in this book the manner of discussing the biochemical disturbances in various diseases is so well and clearly done that the clinician will have little difficulty in understanding it. In the chapter on "Diseases of the Blood" the anemias are discussed and defined with elaboration upon the various types of clinical conditions associated with anemia, such as hemolytic anemia, hemolytic jaundice, sickle-cell anemia, paroxysmal hemoglobinuria, etc., as well as the other better known forms of anemia. Metabolism in anemia is also discussed, as is the blood chemistry. In each way the biochemical aspects of different systems of the body are discussed, such as diseases of the heart, the respiratory tract, the kidneys, the urinary tract, the digestive tract, and so on. Also application of biochemistry to obstetrical and gynecological conditions and the biochemical aspects of neurological and psychiatric disorders are discussed. The book is characterized by its distinct clinical application which is of inestimable value to the clinician and also by the splendid bibliography which follows each chapter.